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E. H. B.

THE TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY

OF EDINBURGH

VOL. XX.—NEW SERIES

SESSION 1900-1901

1901

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PREFACE

THE present Volume is the *Twentieth* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session Extra Meetings were held for the exhibition of Patients, Pathological Specimens, Instruments, etc. It is hoped that such Meetings will materially increase the usefulness of the Society.

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

WILLIAM CRAIG,
Editor.

September 1901.

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	Dr Lewis C. Bruce, The Asylum, Murthly, Perthshire,	1895
	Dr A. H. Buchan, 42 Heriot Row,	1899
	Dr Buist, 1 Clifton Terrace,	1877
	Dr Wm. Hartley Bunting, 74 Bruntsfield Place,	1900
35	Dr T. M. Burn-Murdoch, 14 Charlotte Square,	1886
	Dr Cadell, 22 Ainslie Place,	1870
	Francis M. Caird, Esq., 13 Charlotte Square,	1883
	Dr H. L. Calder, 60 Leith Walk, Leith,	1884
	Dr James Cameron, 13 Fettes Row,	1895
40	Dr Edward Carmichael, 21 Abercromby Place,	1887
	Dr J. Carmichael, 22 Northumberland Street,	1870
	Dr William Carmichael, 25 Braid Road,	1900
	Dr D. A. Carruthers, 12 Rose Terrace, Perth,	1901
	C. W. Cathcart, Esq., 8 Randolph Crescent,	1883
45	Dr J. G. Cattanach, 3 Alvanley Terrace,	1895
	Dr T. F. S. Caverhill, 16 Randolph Crescent,	1883
	Dr George L. Chiene, 26 Charlotte Square,	1901
	Professor John Chiene, C.B., 26 Charlotte Square,	1867
	Dr J. A. Clark, 4 Cambridge Street,	1893
50	Dr Church, 36 George Square,	1876
	Dr Clouston, Tipperlinn House, Morningside Place,	1861
	J. M. Cotterill, Esq., 24 Manor Place, *Secretary*,	1878
	Dr John Craig, 71 Bruntsfield Place,	1900
	Dr William Craig, 71 Bruntsfield Place,	1869
55	Dr Halliday Croom, 25 Charlotte Square,	1870
	Dr G. Matheson Cullen, 48 Lauriston Place,	1892
	Dr A. S. Cumming, 18 Ainslie Place,	1884
	Dr John Cumming, 20 Gilmore Place,	1894
	Dr R. J. B. Cunynghame, 18 Rothesay Place,	1868
60	Dr T. B. Darling, 13 Merchiston Place,	1887
	Dr G. D. Darlington, 10 Ardmillan Terrace,	1898
	Dr M. Dewar, 24 Lauriston Place,	1885
	Dr J. T. Dickie, 37 Lauriston Place,	1900
	Dr George Dickson, 9 India Street,	1884
65	Dr George Dickson, 14 Ardmillan Terrace,	1901
	Dr C. E. Douglas, Cupar-Fife,	1894
	Dr Kenneth M. Douglas, 19 Chester Street,	1888
	Dr J. W. Dowden, 22 Melville Street,	1893
	Dr W. B. Drummond, 9 Brougham Place,	1900

		Date of Admission.
70	Dr John A. H. Duncan, 32 Morningside Drive,	1895
	Dr Kirk Duncanson, 22 Drumsheugh Gardens,	1871
	Dr H. M. Dunlop, 20 Abercromby Place, *Secretary*,	1883
	Dr J. C. Dunlop, 24 Stafford Street,	1892
	Dr J. Dunsmure, 53 Queen Street, *Vice-President*,	1872
75	Dr John Eason, 196 Newhaven Road,	1900
	Dr C. C. Easterbrook, Craig House, Morningside,	1899
	Dr William Elder, 4 John's Place, Leith,	1892
	Dr J. Haig Ferguson, 25 Rutland Street,	1885
	Dr W. A. Finlay, 50 Trinity Road,	1875
80	Dr R. A. Fleming, 10 Chester Street,	1890
	Dr A. Dingwall Fordyce, 34 Great King Street,	1901
	Dr Foulis, 34 Heriot Row,	1875
	Dr J. S. Fowler, 68 Northumberland Street,	1895
	Dr John Fraser, 19 Strathearn Road,	1878
85	Professor Thomas R. Fraser, 13 Drumsheugh Gardens,	1865
	Dr F. Gardiner, 40 Rankeillor Street,	1900
	Dr Garland, 53 Charlotte Street, Leith,	1873
	Dr G. A. Gibson, 3 Drumsheugh Gardens,	1880
	Dr A. B. Giles, 4 Palmerston Place,	1893
90	Dr A. Lockhart Gillespie, 12 Walker Street,	1891
	Dr D. J. Graham, 28 Gilmore Place,	1897
	Dr F. M. Graham, 16 Mayfield Gardens,	1894
	Dr J. Taylor Grant, 3 Manor Place,	1898
	Dr J. Allan Gray, 107 Ferry Road,	1879
95	Dr T. Paul Gray, 6 St Catherine's Place,	1899
	Professor Greenfield, 7 Heriot Row,	1886
	Dr David Greig, 25 Tay Street, Dundee,	1892
	Dr G. L. Gulland, 6 Alva Street,	1888
	Dr A. C. Guthrie, 171 Constitution Street, Leith,	1890
100	Dr William Guy, 11 Wemyss Place,	1890
	Dr William Haldane, Viewforth, Bridge of Allan,	1889
	Dr D. Berry Hart, 29 Charlotte Square,	1886
	Dr James Harvey, 7 Blenheim Place,	1893
	Dr Henry Hay, 19 Nelson Street,	1884
105	J. W. B. Hodsdon, Esq., 6 Chester Street,	1883
	Dr George Hunter, 33 Palmerston Place,	1876
	Dr J. Hutcheson, 44 Moray Place,	1890
	Dr Robert Inch, Gorebridge,	1887
	Dr R. W. Inkster, 31 Windsor Street,	1895
110	Dr Alex. James, 10 Melville Crescent,	1877
	Dr Allan Jamieson, 35 Charlotte Square,	1876
	Dr Hugh Jamieson, 13 Rutland Street,	1889
	Dr James Jamieson, 43 George Square,	1877
	Dr J. Boyd Jamieson, 43 George Square,	1900
115	Dr G. M. Johnston, 7 Wellington Place, Leith,	1888
	Dr R. J. Johnston, 1 Buccleuch Place,	1897
	Dr R. M'Kenzie Johnston, 2 Drumsheugh Gardens,	1883
	Dr C. Kennedy, 5 Salisbury Road,	1886
	Dr C. B. Ker, City Fever Hospital,	1894
120	Dr George Kerr, 6 St Colme Street,	1885
	Dr J. H. A. Laing, 11 Melville Street,	1889
	Dr H. G. Langwell, 5 Hermitage Place, Leith,	1899
	Dr David Lawson, Nordrach-on-Dee, Banchory,	1901
	Dr H. A. Leebody, Corstorphine,	1899
125	Dr Linton, 60 George Square,	1863
	Dr Harvey Littlejohn, 1 Atholl Crescent, *Treasurer*,	1889
	Professor Sir Henry D. Littlejohn, 24 Royal Circus,	1853
	Dr G. R. Livingston, 35 George Street, Dumfries	1901
	Dr T. Davy Luke, 95 Shandwick Place,	1900
130	Dr R. A. Lundie, 55A Grange Road,	1883
	Dr D. C. A. M'Allum, 21 Alva Street,	1900

		Date of Admission.
	Dr P. M'Bride, 16 Chester Street,	1879
	Dr W. A. M'Cutchan, County and City Asylum, Burghill, Hereford,	1897
	Dr K. N. Macdonald, 21 Clarendon Crescent,	1880
135	Dr Stuart M'Donald, 3 Lonsdale Terrace,	1901
	Dr William C. M'Ewan, Prestonpans,	1892
	John M'Gibbon, Esq., 55 Queen Street,	1868
	Dr John M'Gibbon, Junr., 55 Queen Street,	1899
	Dr MacGillivray, 15 Charlotte Square,	1877
140	Dr G. Mackay, 74 Bruntsfield Place,	1878
	Dr George Mackay, 20 Drumsheugh Gardens,	1887
	Dr Robert Mackenzie, Napier, Nairn,	1893
	Dr W. Leslie Mackenzie, 1 Stirling Road, Trinty,	1895
	Dr William A. Mackintosh, 3 Park Terrace, Stirling,	1896
145	Dr J. Shaw M'Laren, 14 Walker Street,	1887
	Dr P. Hume Maclaren, 1 Drumsheugh Gardens,	1868
	Dr Malcolm M'Larty, 7 Bellevue Place,	1900
	Dr John Macmillan, 48 George Square,	1897
	Dr John Macpherson, 8 Darnaway Street,	1891
150	Dr John MacRae, Lynwood, Murrayfield,	1893
	Dr Edwin Matthew, 4 John's Place, Leith,	1900
	Dr D. Menzies, 20 Rutland Square,	1878
	Dr A. Miles, 1 George Square,	1892
	Dr J. Mill, 26 N. Fort Street, Leith,	1886
155	A. G. Miller, Esq., 7 Coates Crescent, *President*,	1867
	Dr W. H. Miller, 51 Northumberland Street,	1895
	Dr D. Milligan, 11 Palmerston Place,	1885
	Sir Arthur Mitchell, 34 Drummond Place,	1859
	Dr J. E. Moorhouse, 1 Glebe Avenue, Stirling,	1901
160	Dr James Mowat, 24 Minto Street	1893
	Dr John Mowat, 6 Buccleuch Place,	1885
	Dr Claud Muirhead, 30 Charlotte Square,	1866
	Dr R. Milne Murray, 11 Chester Street,	1884
	Dr H. Oliphant Nicholson, 20 Manor Place,	1900
165	Dr John Orr, 1 Rillbank Crescent,	1895
	Dr W. Basil Orr, 13 Braid Road,	1893
	Dr G. Keppie Paterson, 19 Albany Street,	1889
	Dr J. V. Paterson, 5 Castle Terrace,	1900
	Dr D. Noël Paton, 22 Lynedoch Place,	1885
170	Dr H. A. Peddie, 24 Palmerston Place,	1893
	Dr R. W. Philip, 45 Charlotte Square,	1883
	Dr Playfair, 5 Melville Crescent,	1874
	Dr T. Proudfoot, 30 Lauriston Place,	1889
	Dr R. B. Purves, 17 Walker Street,	1899
175	Dr Harry Rainy, 16 Great Stuart Street,	1896
	Dr Rattray, Portobello,	1874
	Dr James Ritchie, 22 Charlotte Square,	1873
	Dr R. Peel Ritchie, 1 Melville Crescent,	1862
	Dr W. T. Ritchie, 9 Atholl Place,	1900
180	Dr Argyll Robertson, 18 Charlotte Square,	1861
	Dr G. M. Robertson, The Copse, Larbert,	1890
	Dr William F. Robertson, 20 Dalrymple Crescent,	1893
	Dr W. G. Aitchison Robertson, 26 Minto Street,	1892
	Dr R. M. Ronaldson, 17 Morningside Road,	1895
185	Dr T. R. Ronaldson, 8 Charlotte Square,	1877
	Dr William Russell, 3 Walker Street,	1884
	Dr J. M. Rutherford, Morningside Royal Asylum,	1900
	Dr E. G. Salt, 59 George Square,	1895
	Dr James Scott, 43 Minto Street,	1895
190	Dr Andrew Semple, 10 Forres Street,	1883
	Dr Theodore Shennan, 35 Mayfield Gardens,	1897
	Sir John Sibbald, 18 Great King Street,	1859

		Date of Admission.
	Dr W. C. Sillar, 46 George Square,	1900
	Professor Simpson, 52 Queen Street,	1859
195	Dr G. F. Barbour Simpson, 50 Melville Street,	1900
	Dr Arthur H. H. Sinclair, 5 Walker Street,	1900
	Dr A. A. Scot Skirving, C.M.G., 29 Drummond Place,	1897
	Dr A. T. Sloan, 22 Abercromby Place,	1885
	Dr Andrew Smart, 15 Rutland Square,	1865
200	Dr G. D. Smith, 148 Ferry Road,	1877
	Dr James Smith, 1 Parson's Green Terrace,	1891
	Dr John Smith, 11 Wemyss Place,	1856
	Dr J. Cormack Smith, 9 Brunton Place,	1896
	Dr William Smith, 14 Hartington Gardens,	1890
205	Dr J. W. Steven, 13 Manor Place,	1900
	Dr John Stevens, 2 Shandon Street,	1892
	Dr John Stevenson, 12 Atholl Crescent,	1900
	Dr J. S. Stewart, 15 Merchiston Place,	1885
	Dr Robert Stewart, 42 George Square,	1892
210	Dr William Stewart, 146 Ferry Road, Leith,	1889
	H. J. Stiles, Esq., 8 St Colme Street,	1889
	Dr S. Stirling, 4 Coates Crescent,	1891
	Dr A. Stodart-Walker, 30 Walker Street,	1894
	Dr John Strachan, Dollar,	1867
215	Dr Allan C. Sym, 144 Morningside Road,	1889
	Dr William G. Sym, 20 Alva Street,	1889
	Dr W. Taylor, 12 Melville Street,	1871
	Dr C. C. Teacher, 8 Nile Grove,	1887
	Dr C. H. Thatcher, 8 Melville Crescent,	1876
220	Dr R. Thin, 38 Albany Street,	1890
	Dr J. A. Douglas Thompson, Barmeal, Port-William, Wigtownshire,	1901
	Mr Alexis Thomson, 32 Rutland Square,	1887
	Dr H. Torrancee Thomson, 7 Leopold Place,	1900
	Dr John Thomson, 14 Coates Crescent,	1887
225	Dr T. J. Thyne, 2 Dean Terrace,	1898
	Dr John Tod, 93 Ferry Road, Leith,	1895
	Sir John Batty Tuke, M.P., 20 Charlotte Square,	1864
	Dr F. J. Turnbull, 6 Randolph Place,	1896
	Dr Dawson Turner, 37 George Square,	1890
230	Dr Logan Turner, 20 Coates Crescent,	1892
	Dr Underhill, 8 Coates Crescent, *Vice-President*,	1872
	Dr Norman Walker, 7 Manor Place,	1891
	Mr D. Wallace, C.M.G., 11 Rutland Street,	1887
	Dr David Waterston, 23 Colinton Road,	1900
235	Dr Douglas Chalmers Watson, 22 Coates Crescent	1894
	Dr P. H. Watson, 16 Charlotte Square,	1856
	Dr N. P. Watt, 1 Denham Green Terrace, Trinity,	1898
	Dr A. D. Webster, Belleville Lodge, Blacket Avenue,	1883
	Dr D. A. Welsh, 6 Mardale Crescent,	1900
240	Dr James Wilson, 53 Inverleith Row,	1900
	J. L. Wilson, Esq., 4 Buccleuch Place,	1883
	Dr T. D. Wilson, 10 Newington Road,	1880
	Dr Russell E. Wood, 9 Darnaway Street,	1883
	Dr W. Fraser Wright, Bonnington Mount, Bonnington Ter.,	1894
245	Professor John Wyllie, 44 Charlotte Square,	1868
	Dr P. A. Young, 25 Manor Place,	1870
	Dr R. J. Erskine Young, 24 Comely Bank,	1892

(*b.*) Members Exempted under Rule V. from paying the Annual Subscription.

		Date of Admission.
	Dr D. Aitken, 152 *Packington Street, Islington, London, N.*,	1887
	Dr D. H. Anderson, 131 *Abbey Road, Barrow-in-Furness*,	1887
250	Dr Archibald, *Hazelden, Wimborne Road, Bournemouth*,	1882
	Dr J. A. Armitage, 28 *Waterloo Road South, Wolverhampton*,	1887
	Dr W. Badger, *Penicuik*,	1882
	Dr Edwin Baily, *Oban*,	1883
	Dr Andrew Balfour, *Portobello*,	1874
255	Dr G. W. Balfour, *Westfield, Colinton*,	1847
	Dr J. H. Balfour, *Portobello*,	1881
	Dr Alexander Ballantyne, *Dalkeith*,	1872
	Dr Harold S. Ballantyne, *Dalkeith*,	1899
	Dr Noel D. Bardswell, *Banchory*,	1901
260	Dr W. H. Barrett, 29 *Park Crescent, Hesketh Park, Southport*,	1890
	Dr George T. Beatson, 7 *Woodside Crescent, Glasgow*,	1895
	Surgeon-Captain C. H. Bedford, *Bengal Army, care of W. Watson & Co.*, 28 *Apollo Street, Bombay*,	1889
	Dr Charles Begg, 62 *Pultenery Street, Bath*,	1900
	Dr Benjamin D. C. Bell, *Kirkwall*,	1889
265	Dr G. J. H. Bell, *Bengal Army*,	1884
	Dr G. H. Bentley, *Kirkliston*,	1877
	Dr J. S. Beveridge, *Melton Constable, Norfolk*,	1861
	Dr J. W. Black, 15 *Clarges Street, Piccadilly, London, W.*,	1865
	Dr P. G. Borrowman, *Viewfield, Crieff*,	1893
270	Dr D. G. Braidwood, *Halkirk, Caithness*,	1889
	Dr David Brodie, 68 *Hamilton Road, Highbury Park, London*,	1865
	Dr Andrew Brown, 1 *Bartholomew Road, Kentish Town, London, N.W.*,	1884
	Dr John Brown, *Stockbridge House, Padiham, Lancashire*,	1878
	Dr J. Macdonald Brown, 5 *Lymington Road, West Hampstead,*	
275	*London, N.W.*,	1883
	Dr S. W. Carruthers, 44 *Central Hill, Norwood, London, S.E.*,	1899
	Dr R. D. Clarkson, *Falkirk*,	1896
	Professor Cleland, *The University, Glasgow*,	1864
	Dr A. R. Coldstream, 24 *Lung Arno Nuovo, Florence, Italy*,	1878
	Dr John Connel, *Peebles*,	1876
280	Dr William Craig, *Cowdenbeath, Fife*,	1894
	Dr J. R. Crease, 2 *Ogle Terrace, South Shields*,	1885
	Dr J. Frank Crombie, *North Berwick*,	1897
	Dr P. M. Deas, *Wonford House, Exeter*,	1868
	Dr Archibald Dickson, *Hartree House, Biggar*,	1871
285	Dr A. Halliday Douglas, *Caponflat, Haddington*,	1842
	Dr A. Home Douglas, 5 *Rue de Russie, Nice, France*,	1889
	Dr J. J. Douglas, 42 *Centrall Hill, Upper Norwood, London*,	1891
	Dr W. B. Dow, *Dunfermline*,	1879
	* Dr Thomas Easton, 28 *East Park Terrace, Southampton*,	1894
290	Dr J. W. Eastwood, *Dinsdale Park, Darlington*,	1871
	Dr F. A. Elkins, *The Asylum, Leavesden, near King's Langley*,	1893
	Dr R. W. Felkin, 6 *Crouchhall Road, London, N.*,	1885
	Sir R. B. Finlay, Q.C., M.P., *Middle Temple, London*,	1864
	Dr Simson C. Fowler, *Juniper Green*,	1892
295	Dr F. W. Foxcroft, 33 *Paradise Street, Birmingham*,	1893
	Dr Dyce Fraser, *The White House, Teddington*,	1883
	Dr J. Hosack Fraser, *Fernfield, Bridge of Allan*,	1895
	Professor Gamgee, 8 *Avenue de la Garve, Lausanne, Switzerland*,	1863

		Date of Admission.
	Dr William Gayton, *Bartram Lodge, Fleet Road, Hampstead, London, N.W.*,	1886
300	Dr W. C. Greig, *Otago, New Zealand*,	1884
	Dr George K. Grimmer, *Canada*,	1897
	Dr Groesbeck, *Cincinnati*,	1875
	Dr W. B. T. Gubbin, *Redland Grove, Bristol*,	1893
	Dr John Haddon, *Denholm, Roxburghshire*,	1883
305	Dr Archibald Hall, *Montreal*,	1853
	Professor D. J. Hamilton, *The University, Aberdeen*,	1876
	Dr J. W. Hamp, *Penn Road, Wolverhampton*,	1887
	Dr John Hardie, *Stogursey, near Bridgewater*,	1895
	Dr A. W. Hare, *Newquay, Cornwall*,	1883
310	Dr J. Home-Hay, *Alloa*,	1880
	Professor J. Berry Haycraft, 1 *St Andrew's Place, Cardiff*,	1889
	Dr Stanley Haynes, *Malvern, Worcestershire*,	1864
	Dr R. Dundas Helm, 3 *Alfred Street N., Portland Square, Carlisle*,	1892
	Dr R. E. Horsley, *Jud-Falls, Stoneyhurst, Lancashire*,	1886
315	Dr James Hunter, *St Catherine's, Linlithgow*,	1890
	Dr W. Hunter, 103 *Harley Street, Cavendish Square, London, W.*,	1887
	Dr David Huskie, *Moffat*,	1899
	Dr W. Wotherspoon Ireland, 1 *Victoria Terrace, Musselburgh*,	1883
	Dr J. Carlyle Johnstone, *The Asylum, Melrose*,	1882
320	Dr D. W. Johnston, *P.O. Box 2022, Johannesburg, South Africa*,	1893
	Dr James Johnston, 53 *Princes Square, Bayswater, London, W.*,	1871
	Dr J. Keay, *District Asylum, Inverness*,	1887
	Dr A. J. Keiller, *New Brunswick, Canada*,	1889
	Dr George Keith, *Moidart Cottage, Currie*,	1845
325	Dr Skene Keith, 42 *Charles Street, Berkeley Square, London, W.*,	1885
	Dr W. Scott Lang,	1886
	Dr J. W. Leech, 12 *Eldon Square, Newcastle-on-Tyne*,	1901
	Professor R. F. C. Leith, M.B., *Mason University College, Birmingham*,	1886
	Dr Herbert Littlejohn, *Townhall, Hampstead, London*,	1892
330	Dr Robert Lucas, *Dalkeith*,	1875
	Dr Donald Macaulay, *Jumpers' Gold Mining Co., P.O. Box 1043, Johannesburg*,	1895
	Dr F. R. Macdonald, *Inveraray*,	1860
	Dr W. B. Macdonald, *Dunbar*,	1888
	Dr John A. Macdougall, *Cannes, France*,	1875
335	Professor J. M'Fadyean, 101 *Great Russell Street, London, W.C.*,	1888
	Dr F. Reid Mackay, 27 *York Place, Harrogate*,	1899
	Dr John Mackay, *Aberfeldy*,	1881
	Dr W. B. Mackay, 23 *Castlegate, Berwick-on-Tweed*,	1893
	Professor M'Kendrick, *The University, Glasgow*,	1870
340	Dr James Mackenzie, 68 *Bank Parade, Burnley, Lancashire*,	1894
	Dr T. J. Maclagan, 9 *Cadogan Place, Belgrave Square, London, S.W.*,	1875
	Dr Roderick M'Laren, 23 *Portland Square, Carlisle*,	1882
	Dr S. Macvie, *Chirnside*,	1897
	Dr J. W. Martin, *Burnfoot, Cluden, Dumfries*,	1888
345	Dr A. Matthew, *Corstorphine*,	1882
	Dr Andrew W. Messer, *Lenington, Scotswood-on-Tyne*,	1899
	Dr James Middlemass, *Boro' Asylum, Ryhope, Sunderland*,	1895
	Dr E. A. Mills-Roberts, *Bryn Meurig, Bethesda, Bangor*,	1897
	Dr J. Moolman, *Cape of Good Hope*,	1877

		Date of Admission.
350	Dr E. C. Moore,	1892
	Dr A. E. Morison, *Wellington Road, West Hartlepool,*	1889
	Dr J. Rutherford Morison, 14 *Saville Row, Newcastle-on-Tyne,*	1882
	Professor Robert Muir, *The University, Glasgow,*	1894
	Dr D. G. Macleod Munro, 1 *Oriel Villas, Cheltenham,*	1898
355	Dr J. Ivor Murray, 58 *Addison Mansions, Kensington, London, W.,*	1857
	Dr Andrew Scott Myrtle, *Harrogate,*	1859
	Dr Leith Napier, *The General Hospital, Adelaide, South Australia,*	1879
	Dr T. Goodall Nasmyth, *Cupar-Fife,*	1884
	Dr Ernest F. Neve, *Srinagar, Kashmir, India,*	1884
360	Dr Peter Orphoot, *Greenhythe, North Berwick,*	1865
	Dr T. Wyld Pairman, *H.M. Prison, Lyttelton, New Zealand,*	1884
	Professor Bell Pettigrew, *St Andrews,*	1873
	Dr Alexander Peyer, *Zürich,*	1893
	Dr J. A. Philip, *Rue Victor Hugo, Boulogne-Sur-Mer, France,*	1878
365	Professor W. S. Playfair, 38 *Grosvenor Street, London, W.,*	1857
	Dr J. H. Pringle, 172 *Bath Street, Glasgow,*	1886
	Dr S. Hale Puckle, *Bishop's Castle,*	1885
	Dr J. Maxwell Ross, *St Ruth's, Maxwelltown, Dumfries,*	1882
	Dr J. R. Home Ross, *Burmah,*	1888
370	Dr Joseph C. Ross, *Withington,*	1884
	Dr S. Rumboll, *Cavendish House, Leeds,*	1887
	Dr Thomas Russell, 27A *Westmuir Street, Parkhead, Glasgow,*	1888
	Dr Gordon Sanders, *Cannes, France,*	1892
	Dr F. A. Saunders, *Grahamstown, South Africa,*	1884
375	Dr Thomas R. Scott, *Musselburgh,*	1884
	Dr C. A. E. Sheaf, *Ovalu, Fiji,*	1871
	Dr G. F. Shiels, 229 *George Street, San Francisco,*	1887
	Dr W. H. Shirreff, *Port Fairy, Victoria, Australia,*	1883
	Dr William Simmers, *Denburn, Crail,*	1894
380	Dr T. Skinner, 6 *York Place, Portman Square, London, W.,*	1856
	Dr John Smith, *Brycehall, Kirkcaldy,*	1889
	Dr W. Ramsay Smith, *The General Hospital, Adelaide, South Australia,*	1892
	Dr Van Someren, *Northwood, Middlesex,*	1845
	Dr Somerville, *Galashiels,*	1877
385	Dr A. M. Stalker, 140 *Nethergate, Dundee,*	1893
	Dr Graham Steell, 96 *Moseley Street, Manchester,*	1877
	Professor Stephenson, *University, Aberdeen,*	1861
	Dr Robert Stirling, 4 *Atholl Place, Perth,*	1891
	Professor Ralph Stockman, *The University, Glasgow,*	1891
390	Dr H. R. Storer, *Newport, Rhode Island, U.S.,*	1855
	Dr John Struthers, *Nqamakwe, Transkei, South Africa,*	1895
	Dr C. A. Sturrock, *Dunfermline,*	1894
	Dr J. F. Sturrock, *Arima, Broughty Ferry,*	1887
	Professor J. Symington, *Queen's College, Belfast,*	1878
395	Dr C. Templeman, *Sanitary Office, Bell Street, Dundee,*	1891
	Dr Thin, 63 *Harley Street, Cavendish Square, London, W.,*	1861
	Dr Alexander Thom, *Gowanlea, Peebles,*	1884
	Dr J. Stitt Thomson, *The Mount, Lincoln,*	1877
	Dr J. Batty Tuke, jr., *Balgreen, Murrayfield,*	1886
400	Dr R. S. Turner, *Keith,*	1867
	Dr T. Edgar Underhill, *Dunedin, Burnt Green, Worcestershire,*	1885
	Dr A. R. Urquhart, *Murray House, Perth,*	1893
	Dr Gopal Govind Vatve, *care of H.H. the Rajah of Miraj, Bombay, India,*	1895
	Dr A. B. Whitton, *Aberchirder,*	1886
405	Dr George R. Wilson, *Mavisbank, Polton,*	1892

	Date of Admission.
Dr Oswald G. Wood, *Indian Army*, ...	1886
Professor G. Sims Woodhead, *Pathological Laboratory, New Museums, The University, Cambridge*, . . .	1883
Dr Yellowlees, *Gartnavel Asylum, Glasgow*, . . .	1862
Professor John Young, *The University, Glasgow*, . .	1860

N.B.—*Members are requested to communicate with the Secretaries if they discover any errors or omissions in the List, and also to intimate all changes in their addresses.*

CONTENTS.

I.—ORIGINAL COMMUNICATIONS.

PAGE

(*a.*) PHYSIOLOGICAL.

1. On a Method of Estimating the Interference with the Hepatic Metabolism produced by Drugs. By D. NOËL PATON, M.D., F.R.C.P. Ed., F.R.S.E., Lecturer on Physiology, Surgeons' Hall, and JOHN EASON, M.B., C.M., M.R.C.P. Ed. . . 123

(*b.*) MEDICAL.

(1.) *General Diseases.*

2. The Edinburgh Hospital in South Africa and its Work. By FRANCIS D. BOYD, M.D., F.R.C.P. Ed., Physician to the Edinburgh Hospital in South Africa, Assistant Physician to the Royal Infirmary, and GEORGE L. CHIENE, M.B., C.M., F.R.C.S. Ed., Assistant Surgeon to the Edinburgh Hospital in South Africa 63

3. Some Notes on Alcohol in its Medical and Scientific Aspects. By G. SIMS WOODHEAD, M.A., M.D., Professor of Pathology in the University of Cambridge; with Discussion by Professor T. R. FRASER, Dr T. S. CLOUSTON, and others . . 197

(2.) *Diseases of the Nervous System.*

4. Myasthenia Gravis. By EDWIN BRAMWELL, M.B., M.R.C.P. Lond. and Ed. 116

5. Clinical and Experimental Observations upon General Paralysis. By LEWIS C. BRUCE, M.D., F.R.C.P. Ed., Physician Superintendent, Perth District Asylum, Murthly (*with Charts*). . 171

6. Observations bearing upon the Question of the Pathogenesis of General Paralysis of the Insane. By WILLIAM FORD ROBERTSON, M.D., Pathologist to the Scottish Asylums . 180

(3.) *Diseases of the Circulation.*

7. Arterial Hypertonus and Arterio-Sclerosis: their Relations and Significance. By WILLIAM RUSSELL, M.D., F.R.C.P. Ed., Assistant Physician, Royal Infirmary, Edinburgh (*with Plates*) 132

(*c.*) SURGICAL.

(1.) *General Surgery.*

8. Experiences with the Medical Department of the Army in the South African War. By JOHN CHIENE, C.B., M.D., LL.D., F.R.C.S. Ed., Professor of Surgery in the University of Edinburgh 30

PAGE

9 A Short Note on a case of Multiple Tumours of an Unusual Type. By J. M. COTTERILL, M.B., F.R.C.S. Ed., Surgeon, Edinburgh Royal Infirmary, and Lecturer on Clinical Surgery, and D. A. WELSH, M.D., Pathologist to the Edinburgh Royal Infirmary. 18

(2.) *Affections of the Genital Organs.*

10. On the Operations for Congenitally Misplaced and Undescended Testicle, with Notes of two cases of Congenital Deficiency of the Testicle. By THOMAS ANNANDALE, M.D., F.R.C.S. Lond. and Ed., Regius Professor of Clinical Surgery in the University of Edinburgh 11

(3.) *Diseases of the Abdominal Organs.*

11. Gastric Hæmorrhage and its Surgical Treatment. By A. W. MAYO ROBSON, F.R.C.S., Senior Surgeon to the General Infirmary at Leeds, Emeritus Professor of Surgery in the Yorkshire College of the Victoria University; with Discussion by Dr J. O. AFFLECK, Sir WILLIAM T. GAIRDNER, Professor GREENFIELD, and others 76

12. Stenosis of the Small Intestine after Strangulated Hernia. By ALEXIS THOMSON, M.D., F.R.C.S. Ed., Lecturer on Surgery and Assistant Surgeon, Royal Infirmary, Edinburgh . . 110

13. Operative Interference in cases of Stricture of the Large Intestine. By FRANCIS M. CAIRD, M.B., F.R.C.S. Ed., Senior Assistant Surgeon, Royal Infirmary, Lecturer on Surgery, Edinburgh (*with Plates*) 158

II.—EXHIBITION OF PATIENTS.

(1.) *Illustrating Diseases of the Nervous System.*

1-2. Two Brothers suffering from Friedrich's Hereditary Ataxia. Exhibited by Dr G. A. GIBSON 4

3. A case of Early Tabes. Exhibited by Dr BYROM BRAMWELL . 6

4. A case of Locomotor Ataxia with double extensor response. Exhibited by Dr BYROM BRAMWELL 19

5-7. Three cases of Locomotor Ataxia. Exhibited by Dr BYROM BRAMWELL 45

8. Boy with Paralysis of Gluteus Maximus. Exhibited by Mr H. J. STILES 52

9. A Patient after Laminectomy for Paralysis due to Pressure on Nerve Roots. Exhibited by Professor ANNANDALE and Dr A. BRUCE 53

10. A case of Paralysis of Right Brachial Plexus. Exhibited by Dr ALEXANDER BRUCE 146

11. A case of Acute Disseminated Sclerosis of the Cerebro-Spinal type. Exhibited by Dr ALEXANDER BRUCE . . . 146

12. A peculiar case of Muscular Atrophy. Exhibited by Dr BYROM BRAMWELL 146

13. A case of Syringomyelia. Exhibited by Dr BYROM BRAMWELL . 147

14. A case of Musculo-Spiral Paralysis. Exhibited by Dr ROBERT B. PURVES 155

PAGE

(2.) *Illustrating Diseases of the Skin.*

15. A case of Acne Keloid. Exhibited by Dr ALLAN JAMIESON . 2
16. A case of Tylosis Palmæ et Plantæ. Exhibited by Dr ALLAN JAMIESON 3
17. A probable case of Carcinoma of Skin. Exhibited by Dr ALLAN JAMIESON 4
18. A case of Morphæa. Exhibited by Dr NORMAN WALKER . 48
19. A case of Favus. Exhibited by Dr NORMAN WALKER . . 49
20. A case of Bazin's Disease. Exhibited by Dr NORMAN WALKER 130
21-22. A case of Lupus, and a case of Rodent Ulcer, being treated by X-rays. Exhibited by Dr NORMAN WALKER . . . 131
23. A case of Rodent Ulcer, being treated by X-rays. Exhibited by Mr J. M. COTTERILL 151
24. A case of Ringworm, being treated by X-rays. Exhibited by Dr NORMAN WALKER. 156
25-28. Four cases of Alopecia Areata. Exhibited by Dr NORMAN WALKER 157

(3.) *Illustrating Diseases of Head, Neck and Thorax.*

29. A Patient after removal of Tongue for Cancer. Exhibited by Mr J. SHAW M'LAREN 48
30. A case of Complete Evulsion of the Scalp, showing effects of Skin-Grafting. Exhibited by Mr H. J. STILES . . 50
31. A large Occipital Meningocele. Exhibited by Mr H. J. STILES . 51
32. A Boy who had been trephined for Cerebral Apoplexy. Exhibited by Mr H. J. STILES 150
33. A Patient two months after complete extirpation of the Larynx, for Squamous Epithelioma. Exhibited by Dr M'KENZIE JOHNSTON for Mr ALEXIS THOMSON 20
34. A Boy after removal of a Laryngeal Papilloma. Exhibited by Dr M'KENZIE JOHNSTON 156
35. A Patient suffering from a Tumour of the Left Tonsil, probably a Lympho-Sarcoma. Exhibited by Dr M'KENZIE JOHNSTON . 42
36. A Boy after removal of a large Lymphoma from the Neck. Exhibited by Mr H. J. STILES 21
37. A case of Aneurism of the Transverse and Descending portions of the Thoracic Aorta. Exhibited by Dr BYROM BRAMWELL . 43
38. A Patient suffering from acquired Pulmonary Stenosis. Exhibited by Dr F. D. BOYD 150

(4.) *Illustrating Diseases of the Abdominal Organs.*

39. An obscure case of Regurgitation of Food. Exhibited by Dr H. G. LANGWILL 5
40. A Patient after operation for Perforated Gastric Ulcer. Exhibited by Mr H. J. STILES 49
41-42. Two Patients illustrating certain points in the diagnosis of Gastric Carcinoma. Exhibited by Mr F. M. CAIRD . . 148
43-46. Four cases of Pyloric Obstruction, treated by Gastro-jejunostomy. Exhibited by Mr F. M. CAIRD . . . 61

PAGE

47. An Infant after operation for Acute Intussusception. Exhibited by Mr H. J. STILES 51

48. A Patient after Enterectomy for Multiple Tuberculous Stricture of the Small Intestine. Exhibited by Mr ALEXIS THOMSON . 149

49. A Psoas Abscess in a Girl aged 16. Exhibited by Mr J. SHAW M'LAREN 48

(5.) *Illustrating Diseases of the Extremities.*

50. A case of Elbow-joint affection in a Hæmophilic Patient. Exhibited by Mr J. M. COTTERILL 151

51. A case of Tuberculous Disease of the Wrist. Exhibited by Mr J. SHAW M'LAREN 48

52. A Girl after Amputation through the Hip-Joint. Exhibited by Mr H. J. STILES 22

53. A Patient after removal of the middle two-thirds of the Right Tibia for Central Abscess of the Bone. Exhibited by Mr ALEXIS THOMSON 47

54. A Patient with a Hypertrophic Condition of Integument. Exhibited by Dr EDWARD CARMICHAEL (*with Plate*) . . 147

III.—EXHIBITION OF PATHOLOGICAL SPECIMENS.

(1.) *Illustrating Medico-Legal Cases.*

1. Brain showing Cerebral Hæmorrhage; death by suicide six weeks afterwards. Exhibited by Dr HARVEY LITTLEJOHN . . 59

2. A Cut-Throat. Exhibited by Dr HARVEY LITTLEJOHN . . 59

3. A specimen showing Complete Transverse Rupture of the Small Intestine, also Rupture of the Mesentery, the result of a simple fall. Exhibited by Dr HARVEY LITTLEJOHN . . . 60

4. A Stomach showing Acute Gastritis. Exhibited by Dr HARVEY LITTLEJOHN 60

(2.) *Illustrating Diseases of the Nervous System.*

5. A Tumour of the Right Frontal Lobe of the Brain. Exhibited by Dr BYROM BRAMWELL 9

6-9. Four Brains showing different seats of Cerebral Hæmorrhage. Exhibited by Dr WILLIAM ELDER 24

10. A Large Glioma infiltrating the Right Motor Area and adjacent part of the Cerebral Cortex. Exhibited by Dr BYROM BRAMWELL 27

11. Brain showing Abscesses secondary to an injury to the head. Exhibited by Mr H. J. STILES 58

12. Brain showing complete track of Mauser Bullet. Exhibited by Dr SCOT SKIRVING, C.M.G. 151

13. Brain from a case simulating Ruptured Middle Meningeal Artery. Exhibited by Dr SCOT SKIRVING, C.M.G . . . 152

(3.) *Illustrating Diseases of Head, Neck, and Thorax.*

14. A Skull Cap from a case of Jacksonian Epilepsy. Exhibited by Dr BYROM BRAMWELL 28

PAGE

15. Specimens illustrating Bullet Impacts. Exhibited by Dr SCOT SKIRVING, C.M.G. 153
16. A Larynx from a case of Primary Laryngeal Diphtheria. Exhibited by Dr LOGAN TURNER 154
17. Second, third and fourth Dorsal Vertebræ with a soft degenerated mass growing from their bodies and projecting into the Thorax. Exhibited by Dr WILLIAM ELDER 27
18. A greatly Dilated Heart. Exhibited by Dr ALEXANDER BRUCE for Dr J. O. AFFLECK 106
19. Congenital Fatty Tumour from Axilla of an Infant. Exhibited by Mr H. J. STILES 59

(4.) *Illustrating Diseases of the Abdomen.*

20. Stomach of a Patient eight days after operation for Perforated Gastric Ulcer. Exhibited by Dr SCOT SKIRVING, C.M.G. . 153
21. Intestines from a Girl who had suffered from Idiopathic Distension of the Colon. Exhibited by Mr H. J. STILES . . 154
22. Large Perineal Tumour. Exhibited by Dr SCOT SKIRVING, C.M.G. 153

(5.) *Illustrating Diseases of the Genito-Urinary System.*

23. Two Vesical Calculi. Exhibited by Mr F. M. CAIRD . . 154
24. Vesical Calculus weighing half a pound. Exhibited by Mr F. M. CAIRD 154
25. A large Papilloma from the Bladder. Exhibited by Mr F. M. CAIRD 154
26. Two small Papillomata from the Bladder. Exhibited by Mr F. M. CAIRD 154
27. A Sarcoma of the Testicle of an Infant aged 9 months. Exhibited by Mr H. J. STILES 155
28. A specimen of enlarged Prostate. Exhibited by Mr DAVID WALLACE, C.M.G. 9

(6.) *Illustrating Diseases of the Extremities.*

29. Fragments of Femur from the thigh of a Boer. Exhibited by Dr SCOT SKIRVING, C.M.G. 153
30. Multiple Cartilaginous Loose Bodies from the knee-joint. Exhibited by Mr ALEXIS THOMSON 107
31. A Deformed Shaft of a Tibia. Exhibited by Mr ALEXIS THOMSON 56
32. A recent Pott's (?) Fracture. Exhibited by Mr J. SHAW M'LAREN 57

(7.) *Illustrating Malformations.*

33. A specimen of Double Uterus. Exhibited by Dr HARVEY LITTLEJOHN 60

IV.—EXHIBITION OF MISCELLANEOUS OBJECTS.

(1.) *Mechanical and Surgical Instruments.*

1. A Thermal Bath Cabinet for home use. Exhibited by Dr CHALMERS WATSON 29
2. Schlagintweit's Catheter-Cystoscope. Exhibited by Mr J. SHAW M'LAREN 131

PAGE

3. A Laryngeal Forceps for removal of growths in young children. Exhibited by Dr M'KENZIE JOHNSTON 158
4. A Glass-Barrelled Syringe provided with Nozzles, etc., suitable for irrigating or aspirating. Exhibited by Mr ALEXIS THOMSON . 154

(2.) *Skiagrams, Drawings, Casts, Microscopic Sections, etc.*

5. Radiograph of Fractured Internal Condyle of Humerus. Exhibited by Mr J. SHAW M'LAREN 57
6. Radiograph of Fracture of the Astragalus. Exhibited by Mr J. SHAW M'LAREN 57
7. Röntgen Photograph of Renal Calculi. Exhibited by Dr DAWSON TURNER 108
8. Photographs and microscopic specimens illustrating Progressive Muscular Atrophy, Amyotrophic Lateral Sclerosis, and Glioma of the Pons Varolii. Exhibited by Dr ALEXANDER BRUCE . 154
9. Two Casts showing Congenital Malformation of the Auricle. Exhibited by Dr LOGAN TURNER 154
10. Microscopic specimens from a case of Cervical Tumour. Exhibited by Dr ALLAN JAMIESON 56
11. Microscopic specimens of Actinomyces Fungus. Exhibited by Dr G. L. GULLAND 158

(3.) *Foreign Bodies removed.*

1. Portion ($3\frac{1}{2}$ inches) of Hat-pin removed from the Male Bladder. Exhibited by Mr H. J. STILES 57
2. Twisted Thimble or Sheath of Mauser Bullet. Exhibited by Dr SCOT SKIRVING, C.M.G. 153

V.—DEMONSTRATION.

1-2. Demonstration on the Comparative Pathology of Ataxia; By Dr CHALMERS WATSON (*with Plates*) . . . 62 and 108
3. Demonstration on an Improved Röntgen Apparatus. By Dr DAWSON TURNER 63
4. A series of naked-eye specimens mounted in glycerine jelly illustrating the Pathology of Eye Diseases. Demonstrated by Dr J. V. PATERSON 132
5. A microscopical demonstration of specimens of Actinomyces Fungus. By Dr G. L. GULLAND 158

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TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

FOR SESSION LXXIX., 1900-1901

Meeting I.—November 7th, 1900

MR A. GORDON MILLER, *President, in the Chair*

I. ELECTION OF OFFICE-BEARERS

The following gentlemen were elected office-bearers :— *President*, A. Gordon Miller, Esq. ; *Vice-Presidents*, Dr James Dunsmure, Dr James Andrew, and Dr Charles E. Underhill ; *Council*, Sir John Batty Tuke, Drs J. J. Graham Brown, R. M'Kenzie Johnston, David Wallace, John W. Ballantyne, George Kerr, Kenneth M. Douglas, and H. J. Stiles ; *Treasurer*, Dr Harvey Littlejohn ; *Secretaries*, Mr J. M. Cotterill and Dr H. Melville Dunlop ; *Editor of Transactions*, Dr William Craig.

II. ELECTION OF MEMBERS

The following were elected ordinary members of the Society :—John Stevenson, M.B., C.M., F.R.C.S.Ed. ; 12 Atholl Crescent ; Thomas Davy Luke, M.B., Ch.B., 95 Shandwick Place ; F. Gardiner, M.B., C.M., 40 Rankeillor Street ; Edwin Bramwell, M.B., C.M., M.R.C.P.Ed. & L., 2 Randolph Place ; J. W. Steven, M.B., F.R.C.S.Ed., 13 Manor Place ; H. Oliphant

Nicholson, M.B., 20 Manor Place; John Craig, M.B., Ch.B., Dumfries; W. Carmichael, M.B., C.M., 46 Comiston Road; John Boyd Jamieson, M.B., C.M., F.R.C.S.Ed., 43 George Square; W. C. Sillar, M.B., B.Sc., 46 George Square; W. M'Clarty, M.B., C.M., 7 Bellevue Place; James Wilson, M.B., C.M., 53 Inverleith Row; J. T. Dickie, L.R.C.P. & S.Ed., 37 Lauriston Place; D. A. Welsh, M.D., M.R.C.P.Ed., 6 Mardale Crescent; G. F. Barbour Simpson, M.B., Ch.B., 5 Randolph Crescent; David Waterston, M.D., C.M., F.R.C.S.Ed., 16 Merchiston Terrace; W. H. Bunting, M.D., F.R.C.S.Ed., 74 Bruntsfield Place; A. H. H. Sinclair, M.D., F.R.C.S.Ed., 5 Walker Street; John Eason, M.B., C.M., M.R.C.P.Ed., 196 Newhaven Road; and H. Torrance Thomson, M.D., C.M., 7 Leopold Place.

III. Exhibition of Patients

1. *Dr Allan Jamieson* exhibited—

(*a*) Case of ACNE KELOID.

Mrs H., æt. 40. Sent to Royal Infirmary by Dr Dickson, Lochgelly, in October 1900. Healthy woman, but rather pale and stout. Had seven or eight years since an outbreak of boils on the back of the neck, for which she consulted me, and which completely disappeared for years under the treatment ordered. Three years ago small pustules, then larger boils, from which when ripe thick pus exuded, formed on the nape of the neck. These were accompanied by itching sensations, causing her to scratch and pick them. After lasting eighteen months, a swelling which grew to the size of half a marble came in the centre of the neck, but she cannot tell whether this commenced in the spot where a boil had been or not. This did not increase much in extent till the birth of her ninth and youngest child shortly after. It then extended rapidly and has grown much larger within the last six months. There is now a large irregularly rounded swelling on the nape of the neck encroaching within the hair margin, firm, movable with the skin, pale pink in hue, and somewhat nodulated or uneven on surface. The tumour itself, which projects from the skin about as much as half a good-sized apple, is well defined, but the integument adjoining to the extent of an inch feels thicker than it should be, and there are a few smaller outlying

tumours perceptible at each end of the large one. There are a number of funnel-shaped apertures in the substance of the growth, some are the diameter of an ordinary pin, others twice or three times as large. A probe can be passed into some a distance of half an inch, while from several a few hairs in bunches project. When these are pulled out she experiences a sensation of relief. The only unpleasantness apart from a degree of stiffness in the region, is spontaneous pricking felt at times. The scalp hair is very strong, almost coarse and abundant, quite close up to the tumour.

The nature of both these cases is essentially similar, and corresponds to a well-recognised type of lesion. The origin of ordinary keloid from acne scars is now admitted, and an instance came recently under my notice in a man of thirty who had long suffered from acne of the chest and back, where there were some hundreds of keloid spots about the size of a small shot, which had formed, he assured me, at the acne spots.

A single word as to the name. ACNE KELOID may not be etiologically correct, since, as Unna has shown, there are no true comedones present, but it is the one suggested by Morrant Baker who gave a good account of the disease. It has been adopted by French writers, and is less cumbrous than dermatitis papillaris capillitii that given by Kaposi who first described it. Sycosis keloid has been applied to it, perhaps Pustulo-keloid is the simpler if a change is to be made.

(*b*) TYLOSIS PALMAE ET PLANTAE. The interest in this case of hypertrophy of the horny layer of the palms and soles lies in that it can be traced in various members of the family for at least five generations. T. P., 5 years, was brought to the Royal Infirmary in October 1900. He is a healthy boy and the condition was noticed at birth. The horny layer of the epidermis is thickened into a yellowish, plushlike, somewhat translucent plate on the palms and greater part of the fingers, having an abrupt margin, without any redness beyond. The lines of flexion are deeper than natural, and he feels less acutely than normal. The same condition exists on the soles as far as the heels and front part of the foot are concerned. There are two other children both unaffected. The mother has the like state of skin, of eight in all of her generation three exhibited the malformation. Her mother and seven others out of ten suffered. The great grandfather, still alive and working

at St Andrews has it, and he states that his mother had it likewise, and it is believed that it can be traced still further back. It is closely allied to ichthyosis if not merely a form, and like it probably depends on persistence of the fœtal epitrichium of those regions. The deformity can be kept somewhat in check by rubbing down with pumice-stone. Though the thickened epidermis can be made to exfoliate by such substances as salicylic acid and resorcin, yet it invariably reforms.

(*c*) Case for DIAGNOSIS, probably CARCINOMA OF SKIN.

Mrs S., æt. 38. Sent by Dr Mackenzie, Trinity Road, to Royal Infirmary, 30th October 1900. The growths for which she sought advice were first noticed some nine months since, but she cannot say whether any great change has taken place in them from the time they were originally observed. There are three distinct tumours in the skin on the chest near the axillary end of the left clavicle. The largest is about the size of half a walnut, pinkish in colour, with some dilated venules coursing over it, hard and firm to palpation, well defined and limited to and moveable with the skin. There are two smaller, paler ones quite near, and between and around them is an area of thin, atrophic and glistening tissue, cicatricial in appearance, though there is no history of absorption of other tumours, and not unlike scleroderma in aspect. There is no pain in the lumps nor indeed any abnormal sensation. The axillary or cervical glands are not swollen.

2. Dr G. A. Gibson exhibited two brothers suffering from Friedrich's hereditary ataxy. They belonged to a family of five—four brothers and one sister—of whom the sister and one brother were in good health while the remaining three brothers laboured under the affliction. One of the patients was absolutely confined to bed in a late stage of the disease, the other two were those shown to the Society. In both of them there was comparatively little sensory disturbance ; sensibility to touch, heat, and painful impressions being slightly reduced, sight, hearing, smell and taste were almost normal, but the visual fields were slightly reduced. The organic reflexes were intact, but the cutaneous were diminished. The knee-jerk was lost. Co-ordination was impaired, Romberg's symptom was present, and the estimation of weights was imperfect. The muscular

movements were irregular, and the gait was ataxic. Nystagmus was present, but there was no Argyll-Robertson phenomenon, and no trace of change in the fundus of the eye. The speech was slurring and the writing imperfect. Neither of the patients showed scoliosis, or the "tabetic foot," but in both there was well-marked *pes cavus.*

3. *Dr H. G. Langwill* exhibited an obscure case of REGURGITATION OF FOOD.

G. S., æt. 18 years and 10 months, from North Shields. Complaint, fulness in stomach and inability to eat sufficiently. Vomiting. Duration, three years.

History.—Until three years ago patient took ordinary food without any discomfort. Pain then began in region of stomach, coming on at fifteen minutes *after eating*, and usually ending in vomiting which relieved him. If he was not sick the pain would last about an hour. Never vomited blood nor had melaena. If he *restricted* his diet to fish, pudding, etc., avoiding meat and rich food, he had no discomfort during the *first year.* After a year, however, he began to have a feeling of fulness in stomach *during* eating. This prevented his taking as much as he felt inclined to do. He did not vomit if he took only a small amount of food—not enough to appease his appetite. Pain also occurred if he did not restrict his diet to white foods and milk diets. During this second year of his illness he began to vomit occasionally greenish (bilious-looking) material, *independently of food, e.g.* during the night or before a meal.

During the last year he had not had nearly so much *pain*, but the feeling of swelling and "dead weight" in stomach after even a small meal still is present, and he still vomits if he takes more than a scanty helping of food, or if he does not eat it very slowly. He has lost flesh for the last two years, and has not grown since he was *thirteen.*

Family History.—F. died of heart disease æt. 45.

One brother died in asylum (æt. 11) after fits said to have begun after an eye operation.

Two brothers and three sisters now alive, youngest (10) is as tall as patient.

Condition on admission, height 4.4¾, weight 3.8½ (two years ago weighed about 5½ stones). *Alimentary* system. He takes

his meals very slowly, about three-quarters to one hour to each, *e.g.* as a small cup of beef-tea with crumbled bread and a small helping of pudding. For *breakfast* a small mug of milk and half a slice of bread is all he can manage, but he can take such without pain or vomiting if he rests after every few mouthfuls. He requires to wait about ten minutes after every few mouthfuls to avoid the fulness ending in vomiting. [No heartburn nor waterbrash but some flatulence.] Dry food especially seems to stick about his ensiform cartilage.

Appetite is quite good, but he can't gratify it fully. Tongue clean. Submaxillary glands visible but markedly enlarged.

During dinner and tea especially (unless he eats very slowly) a regurgitation occurs (without retching) of about 2 to 3 oz. of clear fluid, like saliva, with mucus and undigested (recently swallowed) food. It is neutral or alkaline, and contains no HCl.

No swelling or abnormal resistance palpable. No pain in abdomen. Bowels normal, but motions small.

Urine normal.

Treatment.—October 18, stomach tube passed easily for 16 inches from teeth and air pumped in. Attempts at feeding by tube failed as it regurgitated immediately along the tube after about 2 oz. are given.

Weight, October 30, 3.12 lbs.

4. *Dr Byrom Bramwell* exhibited a case of EARLY TABES characterised by recurring attacks of severe vomiting (gastric crises), double Argyll-Robertson pupil, slight lightning pains, slight difficulty in micturition, slight and inconstant thoracic analgesia, in which the knee-jerks were active.

The case was, he said, one of very great interest and rarity.

The patient was an unmarried man, aged 33. Ten years previously he had had gonorrhœa, but denied syphilis. Four and a half years ago he began to suffer from attacks of vomiting; he was at that time seen in consultation by Dr Bramwell. The right pupil was then smaller than the left, but Dr Bramwell had no note as to its reaction to light at that time. During the past four and a half years the attacks of vomiting have recurred with great frequency; they occur on an average every six weeks, and usually last for ten days; the duration of

the shortest attack has been three days and of the longest seven weeks. During the past year the attacks have not been quite so frequent as they were during the first three years of his illness; they have, however, been quite as severe. When the patient was admitted to hospital he was just recovering from the most severe attack he has ever had; it lasted for seven weeks. The attacks of vomiting come on without any apparent cause, dietetic or other. The patient is quite well and does not suffer from any dyspeptic symptoms between the attacks. Diet does not seem to have anything to do with producing the attacks. At the commencement of an attack of vomiting he usually feels cold and often shivers. The attacks are unattended with pain. This is exceptional, for, in the gastric crises of tabes, pain is usually a prominent symptom; some cases have, however, been recorded in which there was no pain. The attacks are characterised by frequent vomiting. The vomited matters are copious; at first the vomited matters contain food, then they become bile-stained, then, though this is not always the case, very dark, sometimes, the patient says, like coffee grounds; and finally colourless and watery, or merely consisting of mucus. The vomited matters are usually very acid, and scald the throat; the patient states that the throat becomes very sore and painful during the attacks. During an attack of vomiting he is unable to retain food on the stomach, and has to be fed by the rectum; he is very thirsty, and drinks a good deal of water during the attacks. During the attacks of vomiting he usually only makes water once in twenty-four hours, and then only a few ounces at a time; on one occasion he only made water once in thirty-six hours. During the attacks the bowels are constipated and the abdomen retracted. In the attacks which Dr Bramwell had personally watched, the pulse frequency was not much altered, and there was no marked change in temperature. He is much pulled down by the attacks, and becomes thin and weak. After the attacks his appetite is voracious.

The stomach and abdomen appear to be perfectly normal. Nothing can be detected there to account for the vomiting. There is absolutely no tenderness on pressure, either during the attacks or during the intervals between the attacks; and, as has been already stated, there are no dyspeptic symptoms.

All sorts of remedies have been tried for the relief of the vomiting. When the patient was first seen, four and a half years ago, large doses of bicarbonate of potassium, with the object of counteracting the excessive acidity of the stomach contents, and the application of a blister both to the pit of the epigastrium and over the course of the vagi nerves on each side of the neck were employed. This treatment seemed for a time to do good; he had no attacks for some months; but the relief was only temporary. Dr Menzies found that the attacks are usually best relieved by a hypodermic injection of morphia. During the last attack antipyrine and phenacetin were given, apparently with benefit; but the attacks vary so much in their severity and duration that it is difficult as yet to speak positively on this point.

The *pupils* were unequal in size, the right being decidedly smaller than the left; the pupils did not react to light but reacted actively to accommodation. During the past year the patient had very occasionally suffered from well-marked *lightning pains* in the lower extremities; on two occasions these pains were so severe that he was unable to sleep all night. During the past year he had had distinct *difficulty in commencing the act of micturition.* The bowels were natural. There was no alteration in the sexual reflexes. *A band of thoracic analgesia* was sometimes present between the 3rd and 6th ribs, but it was slight and variable. The *knee jerks* and *Achilles jerks*, and the *deep reflexes in the upper extremities* were all present and active. The plantar reflex was present and normal; the cremasteric reflex was absent on both sides. Sight was good, the optic discs normal. At the time of the patient's admission to hospital there was marked pseudo-hypotonia apparently due to the muscular exhaustion following the severe attack of vomiting which had immediately preceded his admission.

There could be no doubt that the case was one of tabes and that the attacks of vomiting were gastric crises. It was important to note that the gastric attacks were unattended with pain. Gastric crises were very rare except in tabes, they occasionally occurred in general paralysis of the insane, and also very exceptionally it was said, in disseminated sclerosis and some other conditions.

Professor Leyden of Berlin has described a form of nervous

vomiting, occurring in a periodic manner, without any obvious cause, unassociated with dyspepsia, or with any definite symptoms or signs of organic nervous disease, to which the term *essential nervous vomiting* had been given. But as Professor Dejerine and others had pointed out, it was not improbable that these attacks of periodic nervous vomiting, described by Leyden, were in reality the gastric crises of preataxic tabes. The present case seemed to give support to that view.

IV. Exhibition of Pathological Specimens

1. *Mr David Wallace* exhibited a specimen of enlarged third lobe of the prostate removed by suprapubic cystotomy. The patient was 68 years of age, and had required to use the catheter at intervals for four years. For some weeks previous to the operation catheterisation was daily required and he had lost much blood—the urine was dark red and contained many clots—so that he was rapidly losing strength. The prostate was readily removed without, at the time, much hæmorrhage. The mass removed was as large as a Tangerine orange. After the operation there was no bleeding and the patient left hospital in three weeks: the wound being nearly closed while all the urine was passed per urethram.

2. *Dr Byrom Bramwell* exhibited a TUMOUR OF THE RIGHT FRONTAL LOBE OF THE BRAIN, which presented several features of considerable interest.

The patient, an unmarried man aged 23, was admitted to the Edinburgh Royal Infirmary on 14th April 1897, complaining of headache and loss of vision of nine and a half months' duration. There was no history of syphilis or head injury. The headache had been very severe and was occasionally attended with vomiting. Double neuritis passing into optic atrophy was present. There was no giddiness. The breath was very foul, the tongue furred. The headache was referred to the forehead. There was no tenderness on skull percussion. Vision was entirely lost in the right eye and markedly impaired in the left. There was slight tremor in the hands when the patient held them out in front of him; this was more marked in the right than in the left; the grasping power of right was slightly less than that of the left hand, but the patient was left-

handed. The knee jerks were markedly exaggerated. The memory, mental condition, and intellectual faculties, appeared to be absolutely normal and unaltered. During the patient's stay in hospital he complained on one occasion of a severe pain behind the right eye, and on several occasions the pains were said to be worse on the right side of the forehead than on the left. Drug treatment—iodide of potassium in full doses —failed to produce any benefit. On 12th May 1897, the patient was trephined by Mr Cotterill. The intracranial pressure was very greatly increased; no tumour was found. The operation was done merely as a palliative for the relief of symptoms for there was nothing to show the exact position of the lesion. The effect of the operation was to relieve the headache; no beneficial effect, however, was produced on the eyesight which became completely lost in the left as well as in the right eye. Left-sided hemiplegia followed the operation, and a large hernia cerebri formed at the seat of the trephined wound, the scalp wound being however completely healed. On 31st July, the patient was discharged from hospital.

The patient was seen at his own home on 12th November 1898 and on 29th August 1899. His general health was good. He was completely paralysed on the left side of the body, the arm, face and leg all being affected; the ocular movements were somewhat impaired in all directions, and there was slight nystagmus; he was quite blind; there was little headache; his mental condition seemed quite normal, he talked brightly; his memory and intellect seemed to be unimpaired. An enormous projection was present on the right side of the head, it looked as big as the head itself, it stood out $5\frac{1}{2}''$ from the bone and measured in circumference $20\frac{1}{2}''$. The projection was soft and fluctuating; fluid was constantly escaping from it from a pin-point opening. The patient died at the end of August 1900—three years after the operation.

The *post-mortem* examination was made by Dr Edwin Bramwell. The external projection was a large cyst, the fluid which it contained was clear; a very large tumour, hard and dark in colour, was present in the right frontal lobe. The whole of the right frontal lobe was, practically speaking, destroyed by the new growth which extended backwards towards the motor area and the anterior part of the internal

capsule. The motor cortex and subjacent white matter were extensively altered, apparently as the result of the increased intracranial pressure extruding the brain tissue through the trephined opening in the skull. Communication existed between the lateral ventricle and the external cyst.

The case was of great interest, *firstly*, because of the fact that there were absolutely no mental symptoms or alterations although the tumour had destroyed the whole of the right frontal lobe; *secondly*, because of the absence of any aphasia or speech defects (although the patient was left-handed he wrote with his right hand, and the lesion was of slow and gradual development, hence there was ample time for substitution and compensation); and *thirdly*, because of the enormous size of the extracranial cyst. There could be no doubt that the operation relieved the symptoms and prolonged the life of the patient.

V. Original Communications

1. On the Operations for Congenitally Misplaced and Undescended Testicle, With Notes of Two Cases of Congenital Deficiency of the Testicle

By Thomas Annandale, F.R.C.S. Lond. and Edin., Regius Professor of Clinical Surgery in the University of Edinburgh

Misplaced Testicle

The condition of congenitally misplaced testicle, the result of some abnormal or faulty arrangement of one or more of the attachments of the gubernaculum, is not common. The situations in which such a misplaced testicle have been met with are; on one side of the perineum (most common), and in the region of the saphenous opening in the upper part of the thigh. A third and rare variety has also been described in which the testicle lay in front of the pubis at the root of the penis. When the testicle lies in one of these abnormal situations its presence may give rise to inconvenience, and should it be attacked with inflammation or other diseased conditions, or be complicated with a hernia, still more trouble may be caused.

In connection with operative interference one other fact is

of great importance, and this is that when a testicle remains out of its natural position in the scrotum it degenerates, and its structure alters, so that its functions as a secreter of spermatic fluid are in time quite destroyed. This remark applies not only to the condition of misplaced testicle, but also to that in which a testicle is retained in the inguinal canal or abdomen. The very interesting experiments of Mr Joseph Griffiths, of Cambridge, on dogs, strongly confirms this fact, for he found that when he returned their testicles into the abdominal cavity they quickly degenerated, and soon no trace of the formation or presence of spermatozoa could be found when the animals had been killed.

I have met with two examples of misplaced testicle, both of which were perineal. The first case was one of a child, aged 3 years, which I recorded and figured in the *British Medical Journal* for January 4th, 1879, and was, I believe, the first case in which such a testicle had been successfully replaced and permanently retained in the scrotum. The late Mr Curling, the author of the important work on "Diseases of the Testicle," had previously operated on a similar case, but the organ slipped back into its abnormal position, and the operation therefore was not successful. As the successful result in my case depended upon certain details being carried out in the procedure, details which ever since I have successfully employed in operating also on undescended testicle, I desire to direct attention to them. They are as follows:—

1. An incision is made so as to expose the spermatic cord and its junction with the testicle.

2. The cord and testicle are then drawn out and all attachments fixing the latter in its abnormal position are carefully divided.

3. The corresponding side of the scrotum is opened up with the finger and the testicle placed in it.

4. Two catgut or silk stitches are passed through those portions of the gubernaculum still attached to the testicle where they had been divided, and these same stitches are also passed through the base of the scrotum so as to fix the testicle in its proper position.

5. The opening through which the testicle had passed into the perineum is carefully stitched up by means of subcutaneous catgut sutures.

In this way the testicle is permanently fixed in the scrotum, and I had an opportunity of examining the patient several years after the operation, and found that the testicle had remained in its proper position. In this case the cord was long enough to permit the testicle to be placed in the scrotum, but if in operating in any case of this abnormality, or in the case of an undescended testicle, the cord be found too short, it may be considerably lengthened by a little traction and careful division of cremaster fibres and fascia surrounding the cord, care being taken not to injure the vas deferens or principal blood-vessels.

The second case was similar to the first, but in a patient aged 18. He was not under my care.

The operation for replacing a misplaced testicle is therefore in my opinion to be urged and carried out in early childhood, for a successful procedure such as has been described certainly gives the testicle a chance of developing properly.

Undescended Testicle

When a testicle shows itself outside the abdominal cavity, not having passed properly down into the scrotum, it also is an inconvenience, and may give rise to much trouble should the organ be injured or become diseased. The complication of hernia in such cases is another cause of trouble, and may be attended with risks. When the condition is met with in childhood an attempt to bring down the organ by operation should, I think, be made, as the testicle will not develop properly unless it is placed and retained in the scrotum. It is true that such undescended testicles are usually small, and more or less atrophied, but I say give them their only chance of developing by putting them in their proper place. Should one testicle be properly developed and situated in the scrotum, the operation upon an undescended neighbour need not be so strongly advocated; but when both testicles have not thoroughly descended, or when one is retained in the abdomen and the other has not reached the scrotum, by all means operate at an early period of life.

The complication of hernia with undescended testicle also usually calls for operation, as it is difficult to apply a truss, and such application may still further interfere with the natural descent of the testicle. In the case of an adult I always excise the undescended testicle when operating on the hernia, for the

former is of no use as a testicle, and its removal allows the external ring and inguinal canal to be more thoroughly closed by a "radical" operation on the hernia. In young people I endeavour if possible to bring down the testicle and fix it in the scrotum, and at the same time perform a "radical" operation on the hernia, but if one good and properly-placed testicle were present, I should not be so anxious to preserve the undescended organ.

In operating on cases of undescended testicle I have for many years carried out the details already described, and in my last four cases I have added another detail, and this is to pass the stitches not only through the remains of the gubernaculum and scrotum, but also through the skin on the inner side of the thigh, so as to have a steady traction on the testicle and corresponding side of the scrotum. In one of these cases the patient was a fine lad of 15, whose right testicle was retained in the abdomen, and the left one lay in the inguinal canal close to the external ring. The operation carried out in this case, and in the other two, was most successful, and the first patient is now at a public school playing football and other games.

Cases of Congenitally Deficient Testicle

Congenital deficiency of the testicle is a comparatively rare condition, and I am glad, therefore, to be able to offer brief notes of two cases recently under my care, more particularly as I have by operation obtained the preparations from them, and so have verified their exact condition.

Case I.—M. J., aged 18, was brought to me by his father in order to have my opinion as to some abnormal condition of his left testicle. This condition had existed since birth, and was giving rise to some uneasiness. There was no history of any accident or other trouble, and the patient was a strong and well-developed young gentleman. On examination the left side of the scrotum was seen to be small; at its upper part there could be felt a body much smaller than a natural testicle, soft to the touch, and having an absence of the ordinary testicle sensation when pressed. The corresponding cord felt natural, and the right testicle was placed properly in the scrotum and was well developed. It was decided to explore the body in

the scrotum, which felt somewhat like an atrophied testicle which had not passed sufficiently down. An incision exposing the cord and upper part of the scrotum having been made, the following condition was found. The cord was natural in structure, passed out through the external ring, and ended in the body already referred to, which on examination was not a testicle. The cord was then divided just outside the external ring, its vessels secured, and it, together with the body (the latter of which was firmly attached to the scrotum), was removed. The margins of the external ring were then brought together by two or three catgut sutures. The wound healed rapidly, and this month I had an opportunity of examining the patient and found the parts operated upon quite sound.

CASE II.—J. M., aged 26, admitted into my wards on October 19th of this year, for a condition almost identical with the first case. He was a strong, well-developed young man, and had no history of any accident or trouble, but stated that the left side of the scrotum, and its contents had been present from birth. The right testicle was sound, and in proper position. As in the former case the left side of the scrotum was contracted and small, and contained at its upper part a body somewhat more elongated, but much less in size than an ordinary testicle, and having no proper testicular sensibility. The cord felt natural, and passed out through the external ring, ending in the elongated body, which was fixed to the scrotum. As the condition was causing anxiety to the patient I explored the cord and body, and finding no testicle I excised both, as in the first case. The wound healed quickly, and the patient was going about again on the 5th of this month quite well and with his mind relieved. As the parts removed by operation in both these cases were similar, one description of their exact condition will suffice. For this description I am indebted to Dr Dowden, who was good enough to dissect them for me. The vas deferens was well developed, and could be traced down to the lower part of the body situated in the scrotum. When approaching the body it assumed a series of tortuous curves which terminated in a closed end. There was no trace of any testicle or testicle substance. Around the body, and closely adherent to it, and to the different convolutions of the vas, was a dense firm membrane, the inner surface of which was vascular. The body in the scrotum then in both cases

consisted of convolutions of the vas deferens bound together with the same fibrous bands, and surrounded by a dense membrane which in all probability represented the tunica vaginalis, which was quite shut off from the general peritoneal cavity.

Both these cases must, I think, be considered examples of the rare congenital condition of "absence of the testicle only," this condition being explained by the fact that the testicle and epididymis are independently developed.

Mr Jacobson, in his valuable work on "Diseases of the Male Organs of Generation," states that only four or five of such cases have been recorded. Godard, in his *Recherches sur la Substitution Graisseuse du Rein*, records and figures a case not unlike mine, but in his case an open process or sac of peritoneum passed down along the vas deferens. Mr Jacobson, in his work already referred to, mentions that the following different deficiencies have been met with :—

1. Absence of the testicle only (left one most frequently absent), and this may be unilateral or bilateral.

2. Absence of the testicle, epididymis, and more or less of the vas deferens (most common).

3. Absence of the whole apparatus.

4. Absence of all or part of the excretory apparatus, the testicle being present.

When such deficiencies are unilateral, and the other testicle and its apparatus are natural, the sexual functions and the bodily and mental power are not necessarily interfered with, as exemplified in my two cases, but if bilateral the patients are sterile, and in addition the other genital organs are usually so feebly developed that they are also impotent.

Another Case of Congenitally Misplaced Testicle

Since reading this paper before the Society I have operated upon another interesting case of congenitally misplaced testicle.

Case III.—The patient was a well-developed boy, aged 16, who applied to me in connection with trouble in his right testicle. On examination the right side of the scrotum was small and much contracted ; it contained no testicle or cord, and was quite empty and closed. A small testicle was present at the side of the upper part of the perineum, and its cord could

also be felt passing out through the external ring. At times the testicle and cord disappeared and passed up into the inguinal canal, just below the internal ring. The left testicle was normal and in its proper position. As the right testicle, meanwhile, was giving rise to inconvenience, the boy's father asked me to operate. An incision was therefore made from a point opposite the external ring down to the upper part of the scrotum. At this time the cord and testicle were drawn up into the inguinal canal and so were not exposed by the incision. After a little dissection a sac, much resembling the sac of a hernia, was disclosed, and was carefully separated from surrounding tissues. This sac was opened, and was found to be the tunica vaginalis, which was patent for some distance up the inguinal canal but did not communicate with the general peritoneal cavity. Below the tunica vaginalis ended at the upper part of the scrotum, where it was firmly attached but quite closed. Coming off from this portion of the sac was a diverticulum, which admitted the finger readily and passed down towards the perineum for an inch and a half. It was into this diverticulum that the cord and testicle passed when they descended from the inguinal canal. Having introduced a finger into the inguinal canal and made some traction upon the upper part of the tunica vaginalis, the testicle was easily drawn down, and, its attachments being carefully divided, it was placed in the scrotum and secured there in the method already described. The greater part of the tunica vaginalis and its diverticulum were then excised, a ligature having first been applied above the point of their excision.

The testicle in this case was small but apparently fairly well developed, but the epididymis was not properly developed, the vas deferens ending in a few convolutions at the point of its junction with the testicle.

In this case I passed the stitch securing the testicle to the bottom of the scrotum, through the skin on the inside of the opposite thigh, as I found that this gave traction more directly in the natural line of the cord than when the traction was made from the same side.

In conclusion, operative interference cannot, of course, be of service in such cases of deficiency, but it may, I think, be justifiable in order to remove a condition giving rise to local or mental irritation. If complicated with

hernia, an operation may be necessary in order to relieve the hernia.

REFERENCE.

(1) *Journal of Anatomy and Physiology*, vol. xxvii.

2. A SHORT NOTE ON A CASE OF MULTIPLE TUMOURS OF AN UNUSUAL TYPE

By J. M. COTTERILL, M.B., F.R.C.S.Ed., and D. A. WELSH, M.D.

THE patient, a joiner, had an accident to his wrist seven years ago. Four months later there appeared on the wrist a small growth which ulcerated and spread till it had formed a large, deep, ragged ulcer, which had eaten its way almost half through the wrist, all tissues, bones, tendons and soft parts being eroded by the growth. For over a year he was under treatment in a Glasgow hospital where it was believed to be gummatous in nature, but was not improved by free anti-syphilitic medication. Some improvement followed scraping and cauterisation, but only for a time. The ulcer again enlarged and he was admitted to the Royal Infirmary of Edinburgh in May 1896, where anti-syphilitic remedies again proved useless. Secondary growths in the forearm and epitrochlear gland appeared, the hand was useless, the disease was spreading, and therefore the arm was amputated above the elbow. The wound healed well. In July 1899 the patient returned to the Royal Infirmary with a similar growth on the forehead, and another over the deltoid of the amputated arm. That on the forehead looked like a rodent ulcer, that on the deltoid affected the subcutaneous tissue, and had the naked-eye characters of a small sarcoma or recurrent fibroid. These two growths were freely removed, and the man was well for a year. He returned in July 1900, with a recurrence on the scar of the deltoid, on the fore and little fingers of the left hand, on the great and second toes of the right foot, great toe of left foot, and with ulcers on the right foot and thigh. Those on the fingers were said to have come on after a crush, those on the great toes after ingrowing toe-nails. All these were removed. A beginning growth on the upper lip and columna of the nose was left alone. Thyroid extract did harm. Mercurial inunction combined with potassium iodide was tried, but during this new tumours appeared on the thighs. The case had been seen by

many surgeons and pathologists, and various opinions had been hazarded. Among these were: (1) Tubercle; but there was no family or personal history, nor manifestation in gland, joint, skin, or internal organ, nor bacilli in the ulcers. (2) Epithelioma; against this were the age of the patient, the partial healing of the ulcers after scraping, the sites, the non-involvement of lymphatic glands (except in one instance), the soft edge of the ulcers, and the long duration of seven years. (3) Sarcoma; against this were the chronicity, the partial healing and the absence of internal spreading. (4) Rodent Ulcer; the multiplicity and position of the growths were against this. (5) Syphilis; no gummatous slough ever separated; there were no shotty glands, nor any other sign of congenital or acquired syphilis; there was the absolute therapeutic failure, new tumours appearing during treatment. (6) Farcy; but there was no acute initial attack, no suggestion of this in nose, throat or skin; inoculation of guinea-pigs gave negative results. (7) Mycosis Fungoides; there was absence of the yellow spots like pustulation, as well as the characteristic fungation. The history, too, was against this diagnosis.

Meeting II.—December 5th, 1900

MR A. GORDON MILLER, *President, in the Chair*

I. ELECTION OF MEMBERS

The following were elected ordinary members of the Society:—J. V. Paterson, M.B., C.M., F.R.C.S.Ed., 5 Castle Terrace; D. C. A. M'Allum, M.B., C.M., 31 Alva Street; W. T. Ritchie, M.D., M.R.C.P.Ed., 10 India Street; J. M. Rutherford, M.B., C.M., Royal Asylum; Edwin Matthew, M.B., C.M., Leith; and W. B. Drummond, M.B., C.M., M.R.C.S.Eng., 9 Brougham Place.

II. EXHIBITION OF PATIENTS

1. *Dr Byrom Bramwell* exhibited a case of LOCOMOTOR ATAXIA WITH DOUBLE EXTENSOR RESPONSE.

The patient, a labourer, aged 44, presented all the characteristic symptoms of locomotor ataxia, namely, lightning pains,

loss of knee jerks, double Argyll Robertson pupil, marked ataxia and difficulty in walking, Rombergism, some difficulty in micturition. On examining the plantar reflex, a very marked extensor response of the great toe was found to be present on each foot. This was quite peculiar and exceptional, for in cases of locomotor ataxia the movement of the toes, if any, is flexion not extension. On enquiring particularly into the previous history of the case, it was ascertained that the patient had had two attacks of hemiplegia; one which affected the right side, eleven years previously; and another which affected the left side, two years previously. This entirely explained the double extensor response and showed that the case was not an exception to the rule that in uncomplicated cases of locomotor ataxia, the extensor toe reaction does not occur.

2. *Dr M'Kenzie Johnston* for *Mr Alexis Thomson*, exhibited a patient, two months after complete extirpation of the larynx for SQUAMOUS EPITHELIOMA.

The patient, a man aged 24, had begun to suffer from hoarseness fifteen months ago. Six months ago he became subject to attacks of dyspnœa, and in one of these attacks tracheotomy was performed. Four months ago, Dr Victor Miller of Stockton performed thyrotomy, and cleared out the larynx; the portions of tissue removed were examined by Professor Greenfield and pronounced to be of the nature of squamous epithelioma. The larynx soon filled up again and he was sent to the Royal Infirmary to be under Mr Thomson's care. The region of the larynx was full and tumid, and there was an enlarged gland over the body of the hyoid bone; the epiglottis was so swollen from œdema that it was impossible to inspect the interior of the larynx. On the 1st October, Mr Thomson divided the thyroid cartilage and hyoid bone, scraped out the larynx and evacuated an abscess in relation to its posterior wall, stuffing the cavity with iodoform gauze; the lymphatic gland was removed and was found to be free from epithelioma; the tissues removed from the larynx presented the typical structure of squamous epithelioma. Four days later, the entire larynx was removed along with the anterior wall of the pharynx; the disease had only perforated the box of the larynx at one point on the right side, between the thyroid and cricoid cartilages. Blood was prevented from

passing down the trachea during the operation by keeping the head dependent and by a strip of iodoform gauze wrapped round an ordinary silver tracheotomy tube. The whole wound was left open and lightly packed with gauze; a rubber tube was introduced into the stomach by the wound, but as it made the patient sick, it was removed and he was fed at intervals by a tube passed by way of the mouth. He was out of bed within a week, and was able to swallow everything naturally within a month. He speaks very distinctly but in a subdued whisper.

3. *Mr H. J Stiles* exhibited—

(*a*) A boy, aged 6 years, from whom, seven months ago, he had removed a large simple lymphoma from the right side of the neck. The tumour proved a lobulated mass, extending from the jaw to the clavicle, and, laterally, from the middle of the neck backwards under the the sterno-mastoid to the trapezius. The mass had a peculiar doughy consistence, a condition which, combined with its markedly lobulated character, had led more than one surgeon who saw it to regard it as a multilocular cystic hygroma. The duration of the growth was two years. The opposite side of the neck, and the axilla on the same side, were free from enlarged glands. In spite of the large size of the tumour, Mr Stiles considered it quite possible to remove it, on account of its apparent freedom from adhesion to the main vessels. The incisions employed were one from a little behind the mastoid process to the thyroid cartilage, and a second extending from the middle of this incision downwards obliquely across the sterno-mastoid to the middle of the clavicle. After dissecting the three flaps off the tumour and the sterno-mastoid muscle, the latter was divided and dissected down to the clavicle. The lobulated tumour was then removed in a single mass. No special difficulty was found in freeing the growth from the internal jugular vein and carotid sheath; the spinal accessory nerve, however, which traversed the mass, had to be sacrificed. The loss of blood was not serious. The boy stood the operation well and made a good recovery. To the naked eye, all the glands on section presented the same homogeneous and medullary or brain-like appearance, suggestive of a lympho-sarcoma. There was no caseation. Microscopically it was impossible

to say whether the condition was that of simple lymphoma or lympho-sarcoma. The further clinical history of these cases shows that they are not malignant as there is no recurrence in the true or malignant sense of the term. This boy, for example, has returned to hospital on account of the enlargement since the operation (performed seven months ago), of two or three of the parotid group of lymphatic glands, which were so small that they escaped notice. There has been no recurrence in the immediate neighbourhood of the operation. As to the etiology and pathogeny of the condition we know practically nothing. From the way in which gland after gland becomes enlarged it looks as if we had to do with a chronic infective process, but as yet no micro-organism has been found associated with the condition. Such a multitude of definitely encapsulated lymphoid masses as are seen in this specimen strongly supports the view which he (Mr Stiles) expressed in a paper (The Surgical anatomy of the Breast and Axillary Glands) read before this Society in 1892, viz., that in certain circumstances lymphatic glands may develop after birth from fat lobules, which are more closely related to the lymphatics than usual, just as some fat lobules possess a distinct afferent artery and efferent vein, united by a capillary network occupying the substance of the lobule.

(*b*) A girl, aged 9 years, in whom six months ago Mr Stiles had amputated through the hip-joint and at the same time removed the whole of the acetabular and iliac portions of the innominate bone for tuberculous disease. The patient presented herself at the Children's Hospital two years ago with hip-joint disease, complicated with an anterior abscess. The hip was excised in the usual manner by an anterior incision. She was sent out ten weeks after operation with the limb in plaster. Two months later she was readmitted on account of a collar of tubercular glands extending from ear to ear. In the left submaxillary region were two sinuses, both of which led down to a carious surface upon the jaw, the whole of the left half of which was greatly thickened, and pus discharged freely into the mouth. The glands were excised, and along with them the whole of the left half of the jaw, including the condyle. The bone, which was tubercular, was excised subperiosteally. The parts at the site of this operation are now quite healthy, but little or no new bone has formed.

Unfortunately the disease again broke out in the hip, and twice the parts were freely opened up and the disease as far as possible removed. On the second occasion the acetabulum and adjacent parts of the pubis and ilium were found to be extensively diseased. There were tuberculous pockets and sinuses in various directions, one of the former extending upwards upon the pelvic aspect of the ilium. The patient went from bad to worse, both as regards her local and general condition. The only chance of saving her was to amputate at the hip, and if possible to remove the disease in the innominate bone. Hæmorrhage was arrested by Macewan's manual compression of the abdominal aorta—a method which in children is as efficient as it is simple. The unhealthy raw surface, along with the undermined skin at the site of the old operations, was first freely removed by an elliptical incision, after which the knife was carried with a circular swoop down to the bone at the level of the apex of scarpas triangle. The anterior wound was then deepened down to the bone, the upper part of the femur dissected out, and the limb thus removed. The main vessels were then tied. On opening up the flaps the acetabulum, the horizontal ramus of the pubis and the anterior part of the ilium were found to be so diseased that no good could come of the operation unless they were removed. Accordingly an incision down to the bone was carried backwards from the upper end of the anterior vertical incision parallel to and a little below the iliac crest. The gluteal muscles and the periosteum were rapidly dissected downwards and backwards from the outer surface of the ilium; the ilio-psoas and pectineus were separated from the ilium and pubis and the attachments of the sartorius and rectus divided. The horizontal ramus of the pubis was divided internal to the ilio pectineal eminence, while the ischium was divided below the acetabulum. The ilium was then wrenched away from its sacral attachment. As the patient was by this time suffering severely from shock no attempt was made to ligature the vessels. The large flaps were rapidly brought into apposition with silkworm gut sutures, the patient being sent back to bed as quickly as possible with the artery forceps securing the vessels. Very free drainage was provided. The artery forceps were removed on the following day. Towards evening the patient had recovered from the shock; the pulse,

however, remained irregular and intermittent for several days. She gained strength daily, and was able to leave the hospital fourteen weeks after the operation. She returned to hospital again after an interval of four months; that is to say, about a month ago. There are still two or three deep sinuses which will have to be dealt with. The skiagram, by Dr Rainy, shows that a certain, though small amount of new bone has formed, more particularly in the region of the crest, which, it will be remembered, ossifies by a separate centre. It is interesting to note that the liver which was formerly enlarged, due, no doubt, to waxy disease, is now of normal size.

Mr Stiles observed that he has noticed a similar diminution in the size of the liver in other cases of suppurating tuberculous joint affections where the disease had been removed by operation. The fact is worthy of note as showing the advantage of surgical interference even in very advanced cases of tuberculous bone and joint diseases, provided, of course, that the lungs and other important organs are, as is not infrequently the case, free from tubercle.

III. Exhibition of Specimens

Dr William Elder exhibited—

(*a*) Four brains showing different seats of cerebral hæmorrhage.

Two of these showed hæmorrhage deep in the substance of the cerebral hemisphere and two on or near the surface.

Of the two deep cases, the first was into the internal capsule, the second was into the external capsule, but it had ploughed its way through the temporo-sphenoidal lobe till it reached the surface.

Of the two surface cases, the first was due to a hæmorrhage from the middle meningeal artery and bruise of the tip of the temporo-sphenoidal lobe; the second a hæmorrhage into the cortex of the occipital lobe which had burst through the cortex and spread all over the parietal region and motor areas.

The first three hæmorrhages were from branches of the middle cerebral and the fourth from a branch of the posterior cerebral artery. The third was operated on during life, and blood clot was found; the others were not operated on.

The first was the brain of a man aged 48, who was admitted to Leith Hospital on June 19th 1900, with the following history. Four days before, he had been exerting himself lifting a heavy weight, when he felt as if he had sprained his hand and arm. He did not complain much of it till two days later, when he suddenly felt a feeling of numbness, tingling and weakness in his right hand and leg. This was so bad that he could hardly feed himself. It was only a weakness as he could move his hand, fingers and leg, although not so strongly nor so expertly as usual. He remained in this condition till the morning of admission, when he took severe twitchings of the right arm and leg on three separate occasions. On the first occasion he began to lose consciousness and seems to have been restless and semi-unconscious till admission.

On admission it was found that his right arm and leg were paresed and that he was in a semi-unconscious state. The right arm and leg were more rigid than the left. He improved somewhat till June 25th, six days after admission, when he became more unconscious and more paralysed with cold extremities and lowered temperature. Five days later he died, and at the post-mortem the dura was adherent both to bone and brain. Arachnoid was thickened and sulci full of clear fluid. A small hæmorrhage was found in the internal capsule which had begun in lenticular nucleus and extravasated into internal capsule.

The second was the brain of a man who, eleven hours before his admission to Leith Hospital, had lain down suffering from severe headache and had never wakened. When admitted he was found completely paralysed, there being no difference on the two sides. Pupils were equal, temperature 103°, pulse 120 but twenty minutes afterwards it was 72. Two sides of face were alike, urine slightly albuminous, vessels slightly thickened, heart sounds normal. Eight ounces of blood were taken from his arm soon after admission. Temperature rose to 106° and 107° on two occasions, but was reduced by cold sponging.

At the post-mortem the lateral aspect of the right temporo-sphenoidal lobe was lacerated and a large blood clot occupied a great part of the interior of the temporo-sphenoidal lobe. Some of it was extravasated over the surface of the temporal lobe and marginal gyrus. The bleeding had taken its origin

from the external capsule and had ploughed its way through the temporo-sphenoidal lobe. The pressure had pushed the internal capsule, the corpus striatum, and optic thalamus, and the right ventricle towards the left side, so that the whole left hemisphere was squashed from the pressure and the longitudinal fissure was pushed towards the left side. The case is an illustration of an unilateral lesion producing bilateral paralysis from the size of the blood clot. His kidneys showed subacute interstitial nephritis.

The third brain was from a man aged 41, who was admitted to Leith Hospital on June 9th 1900, in a semi-comatose condition. The night previously he had gone out quite well at 7 o'clock, but at 11.30 his wife met him on the street going home intoxicated. He complained of a bad headache and said he had been kicked. He lay down on bed but became sick, and tossed about a good deal through the night. In the morning he still complained of his head and soon became unconscious. He remained in the same condition all day, and was admitted at night. When admitted he was semi-conscious and had rigidity of the left arm and leg which he moved very little. The pupils were contracted and equal, temperature 101°, and pulse 70 and irregular, but half-an-hour afterwards it was 132. Breathing not stertorous. There was a slight bruise over the right side of the head below the zygoma. Urine normal.

After consultation with Mr Miles an operation was decided on, and a trephine opening was made over the arm and face areas on the right side. A subdural hæmorrhage was found, the wound was enlarged downwards and a large clot with some lacerated brain tissue was scooped out. No fracture could be made out. He improved very little after the operation and died on the fourth day.

At the post-mortem there was found a fracture of the petrous part of the temporal bone and a spicule of bone was sticking in the middle meningeal artery low down. There was laceration of the anterior one-third of the temporo-sphenoidal lobe with a blood clot over the lacerated region.

The fourth was a brain shown to this Society on a previous occasion, and now shown to contrast with the three previous cases. This case had fallen down insensible, and died next

day. He had very little paralysis and little difference between the two sides.

At the post-mortem examination, it was found that a hæmorrhage had taken place from a branch of the posterior cerebral artery into the cortical substance of the occipital lobe near the tip, and had ruptured on to the surface and spread over the parietal region and down to the base in a thin layer.

(*b*) Several of the dorsal vertebræ from an old man who, about six months before his death began to complain of persistent, excruciating pain in his left side over the left mammary region, and sometimes running round to the back. Medicines hardly ever gave him relief. Periodic attacks of persistent hiccough were also a feature in the case. He occasionally got a little better both of his pain and his hiccough, but about a month before his death he became much worse and got more feeble. Suddenly, he lost power in his legs, the left leg a little more markedly and slightly earlier than the right, and in three days he was totally paralysed in both legs, with exaggerated reflexes. Sensation was also lost below the level of the nipples. The arms were not affected. The diagnosis was made of a tumour pressing on the spinal cord, probably growing from the vertebræ at the level of the second or third dorsal vertebræ, which tumour had involved the intercostal nerves on the left side for some months before invading the cord.

At the post-mortem, a soft degenerated mass was found growing from the bodies of the second, third, and fourth dorsal vertebræ projecting into the thorax towards the left of the middle line. The bodies of the vertebræ involved were soft and spongy with pus in interstices of the cancellated tissue. This soft cancellated mass had reduced the size of the spinal canal, so that about the third dorsal vertebra the cord was pressed on, and at this part there was distinct congestion both of the meninges and the cord itself. The nature of the tumour on account of the degenerative changes which had taken place in it could not definitely be made out.

Dr Byrom Bramwell exhibited—

(*a*) A LARGE GLIOMA INFILTRATING THE RIGHT MOTOR AREA AND ADJACENT PART OF THE CEREBRAL CORTEX.

The patient was a sailor, aged 24, unmarried. He was first admitted to the Edinburgh Royal Infirmary on 1st

February 1899, suffering from epileptiform attacks. These attacks were for the most part Jacksonian in character. The spasms commenced either in the left side of the face or the left hand. The patient was discharged in March and readmitted on 26th September 1899, much *in statu quo*. As no decided improvement occurred under iodide and bromide of potassium, the patient was trephined by Mr Cotterill, the condition having been diagnosed by Dr Bramwell as a gliomatous tumour of the brain. Nothing was found at the operation, which was performed on 12th December 1899. The patient made a good recovery from the operation but continued to have fits. He was discharged from hospital on 10th January 1900. He presented himself again on 9th October 1900, very much worse. For a month he had suffered from severe headache and vomiting. Double optic neuritis was now present, and a cerebral hernia of some size projected at the seat of operation. He died suddenly in a fit a few days after his admission.

On post-mortem examination, an extensive area of the motor cortex and of the subcortical tissue was found to be infiltrated with gliomatous tissue.

The case was an excellent illustration of those cases of intracranial tumour in which there are well marked "localising" symptoms, but no general symptoms. Until the later stages of the case, this patient did not complain of headache or vomiting, and during the first year that he was under observation, there was no optic neuritis.

An interesting feature of the case was that although the brain tissue looked normal, when exposed at the time of the operation, muscular contractions could not be obtained by the application of the faradic current to the motor cortex.

(*b*) A SKULL CAP FROM A CASE WHICH IN MANY RESPECTS WAS VERY SIMILAR TO THE LAST ONE.

The patient, a man aged 54, suffered from very numerous attacks of Jacksonian epilepsy. In this, as in the last case, there were no "general symptoms"—no headache, vomiting or optic neuritis; but well marked localising symptoms; the fits began in the left arm. The patient was trephined over the right motor area (arm centre). The brain appeared to be

normal to the naked eye, but did not respond to Faradic current. The patient died suddenly in a fit a few days after his discharge from hospital.

On post-mortem examination, a glioma was found $1\frac{1}{2}''$ from the surface immediately below the seat of operation.

The skull cap was an interesting specimen, and showed the marvellous results which could, in some cases, be obtained by antiseptic surgery; the stitches in the dura, three months after the operation, were as fresh as if they had been put in the day before.

IV. Exhibition of Apparatus

Dr Chalmers Watson exhibited a thermal bath cabinet for home use.

The apparatus consists in a portable folding box $3\frac{1}{2}$ feet high by $2\frac{1}{2}$ by $2\frac{1}{2}$ feet across, made of india-rubber, with a roof arranged to fold over on to the neck and shoulders of the patient, who is seated on a suitable stool within the cabinet. The heating apparatus consists in a specially constructed methylated spirit lamp (in which pure spirit only must be used), provided with a small circular trough in which is placed hot water when a vapour bath is desired. The temperature of the dry bath varies from 100 to 200°, and the vapour bath reaches the maximum temperature of 125°. When desired the temperature is graduated by slightly opening the flaps of the roof. A vapour bath is generally to be preferred, and should be taken in a room of suitable temperature, preferably late at night, an hour before retiring. The usual accessories, such as cold water for sipping, cold cloth to forehead or head, and the like, are arranged as in a major establishment. Facilities are also arranged for sponging the body with cold water at the completion of the bath, after the excess of perspiration has been removed.

The physiological action of the bath is exactly that familiar to and described by Balneologists, and the general feeling of well-being after it is a delicious one. Some idea of its potency might be gauged by the fact that the exhibitor had in several instances seen a loss of weight of exactly half a pound result from a vapour bath of twenty minutes' duration. This fact at the same time indicated that great caution was necessary in arranging the details of the bath for medical purposes. This

was specially true of stout subjects in whom the cardio-vascular disturbance might be profound.

There was no doubt that, apart from its value to healthy people, such an apparatus was calculated to be of considerable benefit in a wide group of medical derangements.

This was not the only form of bath cabinet in the market, but it was the only one of which he had any practical knowledge. Neither was it by any means perfectly new, as the exhibitor had seen cases where patients had used the bath on their own initiative for a considerable time with, as they asserted, very beneficial results.

V. ORIGINAL COMMUNICATION

EXPERIENCES WITH THE MEDICAL DEPARTMENT OF THE ARMY IN THE SOUTH AFRICAN WAR

By JOHN CHIENE, F.R.C.S.Ed., Professor of Surgery in the University of Edinburgh

IN bringing before the Society my experiences in S. Africa, I must keep in mind that my stay there was divided into two distinct periods, from the beginning of April until the end of June, from the end of June till the end of August, from Cape Town to Norval's Pont in the first period, from Norval's Pont back to Cape Town in the second period. At first I was simply a Consultant, who advised in individual cases, afterwards the allegations regarding the Army Medical Department had reached Africa, and it was evident that other questions would necessarily occupy my attention.

These questions are still *sub judice*, and cannot yet be discussed. Whatever may be the verdict of the Commission, everyone must allow that the question of Army Medical reform is only a sub-section of the wider question of Army Reform.

1. (*a*) I purpose saying a few words regarding the lessons which I have learned in the hospitals in South Africa. I will speak of these lessons under the following heads:—Ambulance, Anæsthesia, X-rays, Hospital trains and ships, Bullet wounds, Injuries of the Head, Injuries of Bloodvessels, Injuries of Thorax and Abdomen. Medical cases—Sanitation, Burning

v. Burying; and then close with a word on the work of the civil surgeon in South Africa.

The four great hospitals in the neighbourhood of Cape Town were Wynberg No. 1, Wynberg No. 2, Rondebosch No. 3, and Woodstock No. 4. Much has been said about Woodstock. It is an old building near Cape Town, lying on the shore, and, like all old buildings in South Africa, it was infested at one time with animal parasites, which gave rise to much discomfort. On my second visit great improvement had taken place in this particular. I never noticed any smell from the town drains, which opened into the bay in the vicinity of the hospital. The drain for surface water was a short one. The drain for sewerage opened three hundred yards from the shore, and the current carried the dejecta away from the hospital, and, judging from the excellent results of the surgical cases which I saw there, I cannot look upon the hospital as an unhealthy one.

The dejecta from the hospital after being mixed with izal was buried in a trench on the shore. I examined these trenches and found no smell whatever.

My time was principally spent in the Wynberg and Rondebosch hospitals, two days a week to each hospital. Both were on excellent hospital sites.

Let me first say a word regarding the conveyance of the soldiers from the trains to the hospitals. As I lived at Wynberg I saw them arrive by train, and I saw them immediately after admission to the hospital. Every one must clearly differentiate between hospital trains, and ordinary trains carrying invalids. In the former the soldiers were most comfortable. In the latter it was very often the reverse. I am fully satisfied that in the future some form of portable cooking stove must be carried in these ordinary trains by means of which simple food may be cooked for the soldiers. You cannot trust for a supply of food at the stopping places. It was often very sad to see how worn and weary the poor fellows were on their arrival. Much was done for them at the Wynberg Station by good kind ladies, who were always in attendance. They must, if possible, in future campaigns, be well fed on the journey.

As regards their conveyance from the trains to the hospital, at Wynberg this was done in ambulance carts. On a smooth

road an ambulance cart is fairly comfortable, but, in my opinion, the M'Cormack-Brook Ambulance litters are certainly more comfortable than ambulance carts, and I think it will be a question for the future if these litters should not take the place of carts in the conveyance of patients from trains to hospitals. I came across a member of the Imperial Yeomanry who had been in both as an injured man, and in his opinion there was no comparison in the relative comforts. We see the same thing in civil life. The greater comfort of the litter as compared with the waggon. These litters were on Rickshaw wheels, with rubber tyres, and worked most smoothly.

A question for the future will arise, Can any improvement be made in ambulance waggons for the removal of patients from the battlefield to the nearest hospital? The Boers used, as a rule, waggonettes and Cape carts.

I have frequently heard our soldiers say that they would rather travel in an ox waggon than in an ambulance cart. Surely some improvement could be made. Possibly an open competition would teach us some lessons as to the best form of ambulance waggons. The essentials are strength, easy turning, an easy motion, and the patients easily reached by the attendant. The stage coaches in South Africa are hung on leather. No doubt they are the results of evolution and the survival of the fittest; and many think that the old-fashioned leather springs are worthy of trial for ambulance purposes. The swing of the carriage must not be too great for fear of sickness analogous to sea-sickness.

Anæsthesia.—Chloroform was the favourite drug. Ether and A.C.E. were in occasional use. I never saw any trouble with any form of anæsthetic. A little struggling, but no sickness at the time, and very little afterwards. Can the sickness in civil life be a nervous condition which does not hold good amongst soldiers in time of war? I am fully satisfied that the soldier accepts without demur any decision which is come to as regards his treatment. He is contented and asks no questions. "All right, I am ready," is his answer. He reminds me of the condition of mind in a patient suffering from shock. Not taking much interest in his own case. I speak of the soldiers that I saw at the base long after their injuries had been received; they seemed still to be suffering from chronic shock if I might use the term. Their nervous

system had received at the time of the accident a shock from which they had not recovered. I can only recall one case of a soldier who refused to be operated upon. How different it would have been in civil life. How often is your advice not taken, possibly because the civil patient has a larger choice of doctors, and goes from one to another, seeking advice until he gets that which agrees with his own or his friends' view of the case. The soldier is alone, has no meddling friends, is trained to obedience, and looks on the doctor as one whom he can trust. The other day Dr Lorans, Medical Officer of Health for Mauritius, was in Edinburgh on a holiday. When I spoke to him of the ready acceptance of the soldier when operative treatment was suggested, and of the ease with which chloroform was taken and the absence of all anxiety during its administration, he told me that he was much struck by both of these things happening after the dreadful typhoon which visited Mauritius in April 1892. In consequence of it many operations were necessary on those who had been injured. The Indian population in the Island, in times of quiet, raised many objections to operations. On this occasion everyone was most amenable and no objections were made, and the condition of the patients greatly facilitated the administration of chloroform.

The X-rays.—Great difficulties were often experienced in localising bullets in the spine and pelvis. In the limbs little difficulty arose. In the pelvis a rectal examination will sometimes localise a bullet when the X-rays have been found wanting. I am satisfied that the efficient use of the X-ray apparatus depends very much on the experience of the man who uses it. In some hospitals much want of trust, in others, full belief; in the former inefficient operators, in the latter capable fellows who could localise a bullet without doubt or demur. Mackenzie Davidson's method gained most acceptance.

Another thing which was very evident in South Africa was the distinct division of patients into two great classes. Those who were anxious to go home, those who wished to go back and fight. The first were all septic cases, the second aseptic ones. Dysentery is the disease which most takes the heart out of a man. In the hospitals I saw no one who had not a strong opinion one way or another.

Hospital Trains and Ships.—I am very anxious that my hearers should fully understand the great value of hospital

trains and hospital ships. In Natal the absence of corridor carriages rendered the Princess Christian's hospital train of much value. Most fortunately it was ready just in time to cross the Tugela and bring the sick after the relief of Ladysmith. Colonel Forrester, educated in Edinburgh, was then in charge. Shortly afterwards he was seized with typhoid fever, which occasioned his death.

In Cape Colony there were four trains with every comfort; two of them under the care of Edinburgh men, Major Simpson and Captain Fleming, D.S.O. The good that these trains did is incalculable. Kipling has written of them. I wish his articles which appeared in the *Cape Times* and in the London papers could be reprinted in a separate form. To my mind they gave the best idea of the true condition of affairs.

As regards the hospital ships I had no experience of those fitted up at home, but I saw two of the sick ships fitted up in Durban, the *Lismore Castle* and the *Simla*, and I am sure nothing could be better. Every comfort and every convenience, and it is especially interesting to note that as regards the Durban ships they were fitted up by naval officers, with no special experience, assisted by Major MacCormack. I have heard it said that when these ships went home they were the envy of the skilled artificers who had fitted up the hospital ships at home. Colonel Gallwey, the energetic head of the Natal Medical Service, looked on these ships as base hospitals. They contained 1660 beds, and were a great help in relieving the congestion in the hospitals on the lines of communication. When I mention Colonel Gallwey's name I cannot but recall the valuable work done by the Indian Bearer Company inaugurated by him. All spoke well of their bravery and gentleness.

It has to be remembered that the problem in Natal was a much easier one than that in Cape Colony—50,000 men as compared with 150,000; 200 miles of railway as compared with 1000. These two factors alone made all the difference.

Bullet Wounds.—A very interesting question was constantly raised when a soldier was struck at a near range, the wound was worse than when received at a distance. Between 200 and 400 yards a more ragged wound resulted than when the soldier was struck at 1000 or 1500 yards. Possibly the bullet is like a top, at first it spins round a centre, and then after a time it settles down into its true course. In Johannesberg I

saw several cases of severe brain injuries, often very fatal. In one night three were admitted and all died. I see in the paper that these injuries are explained by expanding or explosive bullets. Such bullets were undoubtedly found in the bandoliers, but I think the cases must be divided into two groups, first, those in which the bone and brain are severely injured and torn; second, those in which with a comparatively small bone wound the brain is much torn. In the first, the injury is probably, if no shrapnel is being fired, due to expanding or explosive bullets. In the second, the Mauser bullet may be the cause when fired at a short range.

As regards the poisoning of bullets, I brought home some of the green-coated bullets, and Dr Hugh Marshall, in the Chemical Laboratory, has kindly examined them, and given me the following interesting report:—

Examination of Green-coated Cartridges (Mauser) from Mafeking.—The coating extends from near the tip of the bullet up on to the shoulder of the case. It has not been applied by dipping, but apparently by smearing the molten material backwards and forwards along the cartridge.

The coating is fatty or waxy in appearance. Over the bullet it is of a uniform green colour and opaque, but over the case the colour is irregular, and the yellow colour of the brass is distinctly visible in small patches through the coating; when examined by means of a lens the colouring matter is here seen to be in the form of small spots distributed more or less thickly through a slightly coloured mass.

When the coating over the bullet is carefully scraped off in very thin layers it is at first found to be colourless, forming a white greasy powder. Some distance below the surface the green colouring is reached, continuing right down to the surface of the bullet.

The colourless material melts about 47° C., and is easily saponifiable by alkalies. It has a distinctly acid reaction.

The green-coloured portions when examined chemically give well-marked copper reactions.

The bullets in these cartridges are composed of an outer steel envelope, plated inside and out with an alloy consisting principally, if not entirely, of copper and nickel, and filled with a lead core.

From the facts stated it is evident that the green colouring

matter was not originally present in the coating, but has been derived from the metal. Probably the cartridges were simply coated with suet or tallow for the purpose of lubricating the rifle barrel. In course of time the fatty acids originally present, and which would increase in quantity on keeping, have attacked the underlying metal, forming green-coloured copper salts, which have gradually spread through towards the outer surface of the fatty material. Apparently the copper is more easily abstracted from the copper-nickel plating of the bullet than from the brass of the case.

From the point of view of those against whom the ammunition is employed there can be little difference between such coated cartridges and those which have not been so treated; from one cause or another the coating is fairly certain to be completely removed before the bullet reaches its billet.

HUGH MARSHALL.

UNIVERSITY OF EDINBURGH,
October 10, 1900.

Head Injuries.—My experience of cerebral herniæ before I went to Africa was very unfortunate, cases rarely recovered. In Africa the majority of the cases that I saw were in a fair way to recovery, and in some the recovery was complete. The antiseptic treatment, in my opinion, could not fully account for it. Possibly it was the larger opening in the bone, because my experience has been that if a hernia appears at a comparatively small opening it goes on growing, and the best chance for the patient is to increase the size of the opening in the skull. The intracranial pressure acts like a wedge, and the larger the opening the less the pressure. Dr Louis Irvine, an Edinburgh man, a civil surgeon at Port Napier, Pietermaritzberg, spoke of the good results of Formalin in cases of cerebral hernia. He used solutions varying in strength from 5 per cent. to 25 per cent. These solutions acted by drying up the mass and were at the same time antiseptic.

I saw several cases of severe brain injury followed by recovery. At Wynberg a patient, when lying on his abdomen, was struck by a bullet on the right temporal region, which passed downwards, forwards and inwards, into his mouth, through the hard palate opposite the right canine tooth. He made a good recovery. In Johannesberg I saw a man in whom

the bullet passed transversely across the brain in the frontal region without bad consequences. At Rondebosch I saw a very interesting case, in which a bullet was removed from the left temporal fossa above the base of the Mastoid process. It had been localised by the X-rays. It entered the skull, to the left of the middle line, passing through both occipital lobes. Right hemianopsia resulted. The curious thing about the bullet was that when removed the base of the bullet was next the bone, the apex of the bullet looking in the direction of the line of fire. I remember seeing at Woodstock a Boer prisoner who had lost by a shrapnel accident a large area of his skull. When I saw him the wound was healed, but the bone was not restored, and as he was a middle-aged man there was little hope that an ossific formation would take place. I suggested in order to prevent injury to the pulsating brain, that he should wear a shield of Britannia metal. He smiled and replied, "That he had had enough of that metal."

Injuries of blood-vessels.—I saw several cases of arterio-venous aneurism in the South African hospitals. They occurred in the neck and extremities. Much improvement resulted from proximal ligature of the main artery when the communication was between the carotid artery and internal jugular vein. In the thigh, ligature of the femoral artery did good. In the leg the aneurism as a rule was laid open, and the artery and vein ligatured above and below the opening. As far as I could judge, the cases were more allied to aneurismal varix than varicose aneurism, but there was not the great dilatation of the vein which is commonly described in such cases. Possibly the cases were too recent, and the venous dilatation occurs at a later date. I had no opportunity of making a dissection of any of those cases of arterio-venous aneurism in the neck, but the improvement that took place after proximal ligature of the main artery with the absence of any venous dilatation, made me wonder if the condition could be due to bruising of the artery and vein and subsequent matting with constriction. No one could avoid noting the number of cases of painful neuritis from bruising of the nerve, the cases of neuritis due to adhesion of the nerves to bone after fracture, and the cases of neuritis due to splinters of bullets lodged in the nerve, without thinking that possibly the same thing might have occurred to arteries and veins lying side by

side, and giving rise by matting and constriction to symptoms similar to those met with when there is a communication between the artery and the vein.

One case of arterio-venous aneurism in the neck made a deep impression on me. The bullet had passed through his neck from side to side, the track of the bullet passing behind the trachea and behind or through the oesophagus. There was a pulsating tumour low down in the neck immediately above the clavicle, so low down that nothing but distal ligature of the carotid could be attempted. He developed enteric fever and was invalided home. I heard of him recently in Devonshire jumping a six-foot wall. No operation had been performed.

I may mention a strange coincidence in relation to arterio-venous aneurism. Two brother officers in one regiment were wounded in the same battle. In one the bullet entered on the right side, two inches below the angle of the jaw, the wound of exit being at a corresponding point on the opposite side of the neck. In the other the track of the bullet was the same, the course reversed. In both an arterio-venous aneurism developed near the wound of exit. They were taken off the field in the same ambulance and brought down to Wynberg. Mr Makins operated ligaturing the common carotid in both cases with very decided improvement. In false aneurisms the sac was laid open and the artery tied above and below the bleeding point.

Injuries of Thorax.—In lung injuries I was struck with the rarity with which the patient said that he spat blood at the time of the accident. Apparently the Mauser or Lee-Metford bullet passes through the lung, searing it and preventing hæmorrhage. On the other hand, it must be noted that in many cases there was hœmathorax, probably from the intercostals, and most of the surgeons were agreed that if the blood were slowly absorbed tapping assisted absorption, and there was also general agreement that a rise in temperature was the first indication that absorption had commenced. Possibly this rise is analogous to the rise we see for a day or two after an extensive operation due to absorption of the products of metabolism. Empyœma as a result of lung injuries rarely occurred. I only saw four cases.

I saw several cases of penetrating thoracic wounds in which, after careful examination of the apertures of entrance and exit, I could not but come to the conclusion that the bullet had traversed the heart—I can only express this as my opinion—I earnestly hope that a careful tabulation of all these cases will throw some light on the effects of the Mauser bullet on the viscera—lungs, intestines, heart. In all the cavities I saw wounds which from my former experience would have certainly proved fatal.

Abdominal Injuries.—I had no opportunity of forming an opinion on the question of immediate operation in penetrating wounds of the abdomen. I can only say that I saw several cases in which complete recovery had taken place without operation. It was currently believed that the hungrier a man was the less his risk in an abdominal wound. I saw one case at Kroonstadt in which the wounded bowel had been excised, and I heard of another successful case in Natal. As in the lung, so in the intestine, apparently the Mauser bullet makes so small a wound that neither bleeding nor extravasation occurred. I can explain the recoveries after penetrating wounds in the abdomen in no other way. I saw in Wynberg two very interesting cases of penetrating abdominal wounds, in which a large hematoma formed in the iliac fossa. In one the abdomen was opened and the clot expressed, in the other absorption took place.

In one case the bullet traversed both thoracic and abdominal cavities without evil consequences. It entered above the left clavicle, and was removed from Scarpa's triangle on the right side.

After I left Wynberg, I was brought in contact with a new state of affairs. To a great extent, in Kimberley, Bloemfontein and Kroonstadt, almost entirely in Johannesberg and Pretoria, the sick and wounded were in hotels, churches, and halls, and if there was free ventilation and the rooms were large, they made excellent hospitals, but tents and hospital huts are better than houses. In speaking to the students at the beginning of the session, I referred to what seemed to me a matter of very considerable importance. The purification of these buildings must be very thorough. Many South African friends expressed to me their fear of the coming year, and they anticipate a great outbreak of enteric in the near future. This will be a very

serious affair, looking to the flood of emigrants, new to the country, who will rush north whenever they get the chance. I think there should be no delay in sending out the best incinerators, and compelling the authorities to burn the dejecta. Burying is common in Cape Colony, in the Orange River Colony and Transvaal. In the three large hospitals in Natal, Estcourt, Mooi River and Howick the dejecta were burned. At Johannesberg I made a careful examination of the methods used in burying. I found all the Kaffirs who removed and buried the dejecta in good health, and in a very short time the earth in the trenches in which the burial had taken place resumes its normal appearance. Still I cannot help thinking that burning is infinitely preferable, and I trust that it will soon become general throughout South Africa. There should certainly be incinerators at all the railway stations, to burn the dejecta from the hospital trains.

We will, I trust, hear from Dr Boyd his experiences on typhoid and dysentery, but I may be allowed to say that phlebitis of the common femoral vein was a frequent complication in enteric, delaying recovery. I did not hear of any fatal results from it. Patients with typhoid died from exhaustion or hæmorrhage; rarely from perforation.

There is much difference of opinion as to the merits of typhoid inoculation. It will be some time before the results can be tabulated, because the medical sheets of the soldiers did not accompany them, and no definite data could be obtained, more especially from those who were admitted into hospital in a moribund condition.

There is a fever in the nomenclature of disease used in the army called "Simple continued fever." Many hold that it is aborted typhoid, and at Estcourt Hospital I was informed that five out of six cases reacted to the Widal test. Had these patients been inoculated for typhoid, and was the reaction due to the inoculation?

The Widal reaction test was not in use as a rule in the military hospitals. It is from the civil hospitals that we will hear regarding its value as a diagnostic.

As regards dysentery, I was struck with the great depression of spirits of the patient. The treatment by the free use of sulphate of magnesia, followed by ipecacuanha, was the favourite remedy. Dr Watkins Pitchford, a civil surgeon at

Estcourt, strongly recommended the use of izal along with bismuth in chlorodyne in dysentery. He has described the treatment in the *British Medical Journal*, November 10, 1900. He used it on the suggestion of Dr Crossland, a civil surgeon on duty at Estcourt.

Abscess of the liver sometimes followed dysentery. Those who had most experience of this disease in India advocated posteria aspiration; then if the pus was found, removal of a rib and free drainage. All dwelt on the importance of a very large drainage tube.

Two days ago I received a letter from the Station Hospital, Ladysmith, dated 1st November 1900, portions of which letter, with your permission, I will read, just to indicate the sort of work which falls to the civil surgeon in South Africa:—"I thought you would like to know more of the cases you were consulted about in the surgical ward here early in August. The Ayrshire footballer with dislocation of the internal semilunar cartilage I operated on, finding only a small portion of it turned over, I passed a chromic catgut through it and the capsule of the joint. The wound healed by first intention. The Yeoman with acromegaly was discharged to Howick and invalided home. The orderly with varicose vein was operated on by tying the saphenous vein. He made an excellent recovery, and is regularly on duty. The young scout with locking of the elbow joint was sent to Mooi River Hospital to have an X-ray photograph taken. The night he got there a great storm occurred. All the tents were blown down, and all patients transferred to various hospitals down the line. The most interesting case, I think, was the Grenadier Guard, in whom you diagnosed division from bullet wound of the medial nerve. The day before I intended to operate, I thoroughly overhauled the arm, trying to locate the ends of the divided nerve. In this I failed, but next morning he woke with returned sensation in the hand. I take it the nerve was not divided at all, but had become tied down and strangled in fibrous tissue in the healing track of the bullet, and the manipulation had freed it. Under massage he made rapid progress, and was discharged to Howick with a very useful and still improving hand. I had my best week in South Africa last month. An operation for abscess of the liver from gall stone; iridectomy for glaucoma; radical cure of hernia;

laporotomy for an abscess of the spine after enteric—all doing well."

This letter is from Dr J. B. Rudduck, civil surgeon. Possibly such a letter will best illustrate what seems to be an outstanding fact, viz., that the civil surgeons have been of much service in South Africa.

This paper is chiefly one of impressions. I wish I had been able to keep more notes, but the fact is that, from the day I left Cape Town until I reached Cape Town again, with the exception of a few days at the Edinburgh Hospital, I was constantly on the move, and not placed in favourable circumstances for extensive note-taking.

May I in conclusion express the earnest hope that the medical history of this war will be fully described in the form and with the thoroughness that the great Civil War in America has been dealt with by the Government of the United States. From such a history many most useful lessons will be learnt which will be of the greatest use to all members of our profession, but specially to the Royal Army Medical Corps and to those civil surgeons who may in the future be called on to serve Her Majesty in times of war.

Meeting III.—December 19th, 1900

Mr A. GORDON MILLER, *President, in the Chair*

I. EXHIBITION OF PATIENTS

1. *Dr M'Kenzie Johnston* exhibited a patient suffering from a tumour affecting the left tonsil, probably a LYMPHO-SARCOMA. The patient, a man of 24, healthy in appearance, noticed only about two and a half months before that he had some difficulty in swallowing, and occasionally that solids and liquids were regurgitated through the nose. He never had any pain, and his attention had not prior to this been attracted to his throat, although a month or two earlier friends had noticed a change in his speech. The growth is now rapidly increasing, and extends far beyond the middle line as well as far down into the pharynx. There is no ulceration, though

bleeding is easily produced by touching it with a probe. There is no distinct gland enlargement elsewhere. It is intended at once to remove a portion for examination.

2. *Dr Byrom Bramwell* exhibited—

(*a*) A case of ANEURISM OF THE TRANSVERSE AND DESCENDING PORTIONS OF THE THORACIC AORTA, in which the aneurism had been so completely cured by rest and iodide of potassium that the patient had been able to follow a laborious occupation for twenty-four years.

The patient, a married man, aged 68, was admitted to the Edinburgh Royal Infirmary on 8th November 1900 (on the recommendation of Dr Gulland, who had generously placed the case at Dr Bramwell's disposal), complaining of shortness of breath on exertion, angina-like pains in the chest passing to the left shoulder and down the left arm (which had been removed), cough, spit and difficulty in swallowing.

The history of the case was as follows:—At the age of 20 patient had gonorrhœa, but denied syphilis; at the age of 28 he was discharged from the army, after four years service, on account of disease of the left elbow joint; shortly afterwards the left arm was removed, just below the shoulder, in the Manchester Royal Infirmary; after recovering from the operation, he was employed as a labourer. In the year 1876 he was admitted to the old Edinburgh Royal Infirmary, under the care of Dr G. W. Balfour, complaining of weakness, pain in the chest, both persistent in character and shooting to the left shoulder and down the left arm (which had been removed), and difficulty in swallowing. The condition was diagnosed as aneurism of the aorta. He was treated in the Infirmary for fourteen weeks with rest and iodide of potassium, and was then discharged free from pain and cough, but still complaining of some difficulty in swallowing solid food. After six weeks he returned to work, and for the next twenty-four years—with an interval of eighteen months (when he kept lodgers)—was employed as a hawker, walking twelve, fourteen, or twenty miles a day, and carrying a pack, weighing from twenty to forty pounds, slung on his back after the manner of a knapsack. For the past two years he had been employed as a night-watcher, and in this employment he had been subjected to a good deal of exposure. Two months ago he began to

complain of cough, pain in the chest, and renewed difficulty in swallowing. During the whole of the twenty-four years which had elapsed since the aneurism had originally been treated, he had had some difficulty in swallowing solids (meat).

On admission his height was 5 ft. 7 in., and his weight 8 st. 12 lbs. (he had lost 8 lbs. in weight in the past two months).

Some dulness and crepitations were present over the apex of the left and also at the base of the right lung: the sputum was copious in amount: it did not contain the tubercle bacillus.

The signs of aneurism were as follows:—Slight prominence of the third left costal cartilage; distinct pulsation in the second left interspace; dulness on percussion over the manubrium sterni, and both to the right and to the left of it, but particularly to the left side; loud accentuation of the aortic second sound over the dull area; a slight, soft, systolic murmur in the same position; the right carotid pulse a little larger than the left. At the time of his admission to the hospital the right pupil was slightly larger than the left, but the inequality was not observed later. There was no paralysis of the larynx. During the six weeks that the patient had been in hospital marked improvement in the lung condition occurred, and the patient gained 4½ lbs. in weight; the pain in the chest was greatly relieved, and there was less difficulty in swallowing.

Dr Bramwell thought there could be no reasonable doubt that the case was one of aneurism of the thoracic aorta, and that Dr Balfour's diagnosis had been correct. If this were so, the case was a remarkable example of the beneficial effects of the rest-iodide plan of treatment, which Dr Balfour had done so much to introduce to the profession. He (Dr Bramwell) had seen many cases of aneurism which had been remarkably benefited by the iodide plan of treatment, but he had not met with any case in which the aneurism had been so completely cured by the treatment that the patient had been able to follow a laborious occupation, involving much strain and hard work, for a period of twenty-four years.

Another point of interest in the case was the fact that the angina-like pains were felt by the patient in the fingers of the left hand which had been removed forty years previously.

(*b*) Three cases of LOCOMOTOR ATAXIA in which marked improvement had resulted from the treatment by co-ordinated muscular movements (Fraenkel's method).

Case 1.—A married woman, aged 50, was admitted to the Edinburgh Royal Infirmary, on 13th November 1900, suffering from typical tabes of several years' duration. The patient had suffered for ten years from pains in the legs, said to be rheumatic, and for three years from lightning pains in the legs and lately in the arms; for two years there had been attacks of diarrhœa and abdominal pain, apparently gastric crisis. Two years ago, difficulty in walking and ataxia developed. The knee jerks and Achilles jerks were absent, the arm jerks were not obtained, the jaw jerk was present; the plantar reflex was flexion of the toes; the abdominal reflexes were not obtained; the bladder had been affected, but was now normal; the bowels were normal; the pupils were unequal in size, the right measured 2 mm. and the left 1 mm., they did not contract to light, but reacted to accommodation; Rombergism was marked; there was some hypotonia in the legs; distinct thoracic analgesia was present at the level of the 3rd, 4th and 5th interspaces on the left side; Biernacki's sign was well marked; parasthesia was present; there was some numbness in the feet and legs; there was marked loss of the sense of position of the legs both during and after passive movements; the stereognostic sense was perfect; the sensibility of the muscles was markedly impaired; the gait was highly ataxic, the ataxia was more marked in the left than in the right leg; there was slight ataxia in the arms.

At the time of patient's admission to hospital, she could only walk when supported on one side. As the result of a month's treatment, very great improvement had taken place; the patient was now able to walk without support, to turn easily, the gait now presented very little ataxia. The patient stated that she had not been able to walk so well for two years.

Case 2.—A married woman, aged 42, was admitted to the Edinburgh Royal Infirmary, on 17th November 1900, suffering from typical tabes. The disease had commenced ten years previously with shooting pains in the legs; two years ago, difficulty in walking and ataxia had developed. For fifteen months the patient had been unable to walk without support.

On admission, the knee jerks, Achilles jerks, arm jerks and jaw jerks were all absent; the plantar reflex was present, but sluggish, with flexion of the toes; the abdominal reflexes were active; there was some difficulty in micturition and some constipation; the pupils were equal and myotic, they did not contract to light, and only slightly to accommodation; a band of thoracic analgesia was present; the patient complained of numbness in the feet and legs and occasionally in the hands; the muscular sense was markedly impaired; there was considerable hypotonia; the muscles of the calves were markedly analgesic; Biernacki's sign was well marked.

At the time of her admission to hospital, the patient was unable to stand and walk unless supported on each side; the movements of the legs were wildly ataxic and those of the arms slightly ataxic.

As the result of a month's treatment, great improvement had taken place; the patient was now able to stand and walk with very little support, holding a hand on one side. The patient stated that she had not been able to walk so well for the last fifteen months.

Case 3.—A man, aged 44, was admitted to the Edinburgh Royal Infirmary, on 12th October 1900, suffering from marked tabes. The disease had commenced four years previously with lightning pains in the legs, and diplopia.

On admission, the knee jerks, Achilles jerks and arm jerks were absent; the pupils were unequal, they did not react to light and accommodation; muscular sense was markedly impaired; there was very extensive analgesia; the bladder and rectum were markedly affected, the patient suffered from frequently recurring intestinal crises; trophic lesions of the toes were present; there had been marked loss of weight; the gait was highly characteristic and ataxic.

Under treatment (salicylate of bismuth and rectal feeding) the intestinal crises had disappeared, and the patient had gained strength and weight. For the past month he had been treated by Fraenkel's exercises. His walking power had greatly improved; he stated that he had not walked so well for eight months, and that his general health had not been so good for the past two years.

Dr Bramwell stated that in this case part of the improvement in walking was no doubt due to the increased muscular

strength and the improvement in the general condition, due to the disappearance of the intestinal crises and diarrhœa. This (the improvement in the general health and strength after admission to hospital) was a source of fallacy which must always be taken into account; but, allowing for the improvement due to this cause, both in this and the other cases, he was perfectly convinced that the method of treatment—co-ordinated muscular movements—had produced very marked improvement in all of the three cases shown to the Society. He was indebted to his son, Dr Edwin Bramwell, for his knowledge of the method, and for superintending the details of the treatment.

3. *Mr Alexis Thomson* exhibited a patient in whom he had REMOVED THE MIDDLE TWO-THIRDS OF THE RIGHT TIBIA FOR CENTRAL ABSCESSES OF THE BONE.

The patient, a boy aged 15, was laid up with an illness eight years ago, in the course of which abscesses formed in the lower part of the left thigh, and the right tibia became painful and swollen. The abscesses in the thigh were opened by his doctor, and discharged pus for three months. The tibia became enlarged and curved, but otherwise gave him no further inconvenience.

Without any apparent cause, the disease in the right tibia became active eight months ago. He suffered from pain in the bone every night, so that his sleep was very disturbed. There was no pain during the day. Two months later the skin over the lower half of the shaft of the tibia became red and swollen, and gradually gave way, resulting in three superficial ulcers, with livid, undermined edges. So long as these remained open the nocturnal pain was much relieved, but when they healed the pain relapsed. They healed up and broke down again several times. The ulcers described had no discoverable connection with the subjacent bone. The tibia was much increased in girth, about 1½ inches longer than the left, and curved with the convexity forwards. It was intensely tender on percussion about the middle of the shaft.

The condition was regarded as one of *relapsed staphylococcal osteomyelitis*, dating from the attack of eight years ago, the tibia being in a condition of hyperostosis and sclerosis, as well as lengthened, and probably containing in its interior a cir-

cumscribed (Brodie's) abscess. The bone was too sclerosed to give useful skiagraphs. The operation on July 12th consisted in reflecting the soft parts, including the thick periosteum, as a quadrilateral flap, dividing the shaft of the bone with the saw above and below, so as to remove the central two-thirds. The divided periosteum was stitched with catgut, and the wound closed. The formation of the new shaft was observed in successive radiographs. The newly-formed tibia was more bulky than the same bone on the left side, but was of the exactly same length. He was given liberty to walk on the leg at the end of three months.

4. *Mr John Shaw M'Laren* exhibited—

(*a*) A PSOAS ABSCESS in a girl, aged 16. She had had curvature for years, which latterly had become much worse, and there was considerable pain. A very small incision was made over Poupart's ligament, the pus was allowed to escape. There was then thrown into the abscess cavity several ounces of a 10 per cent. emulsion of iodoform in glycerine, and the wound sutured. The sutures gave way, but there was no pus, and no temperature. The wound healed in six weeks, and the abscess was cured. Thus there was no prolonged operation with scraping, no excision, and yet the result was perfect.

(*b*) A case of TUBERCULOUS DISEASE OF THE WRIST, following tuberculous synovitis twice excised. It was treated by Bier's conjested bandage, and eight injections of iodoform emulsion. Now there was no pain, and there was free movement, though disease was advanced before treatment.

(*c*) A patient who had had CANCER OF THE TONGUE (verified by the microscope), with involvement of the glands under the jaw on both sides. Kocher's incision on each side was made with ligature of the lingual and facial arteries, extirpation of glands, and removal of the whole tongue from the mouth. Though there had been wide involvement, no recurrence had taken place during a period of now more than two years since the operation.

5. *Dr Norman Walker* exhibited—

(*a*) A case of MORPHÆA in a young woman, aged 22. The disease had lasted about eighteen months, and was situated on the forehead and scalp, almost exactly in the middle line.

In the centre was a scar, which was caused by the forceps at the patient's birth.

(*b*) A case of FAVUS, which was only interesting on account of certain facts in the history. The patient was a young man, aged 18, who had never been out of England until two days previously. He had had the disease for ten years. He was one of a family of nine, and no other member of the family was affected. Dr Walker proposed to treat him by exposure to the X-Rays, the effect of which was to cause a rapid falling out of the diseased hairs.

6. *Mr H. J. Stiles* exhibited—

(*a*) A servant, aged 19, who, on January 29th of this year, was operated upon for a PERFORATED GASTRIC ULCER. Mr Stiles said: "I was asked to see the patient by Dr Ballantyne, of Dalkeith, who gave me the following history. The patient had suffered from anæmia and indigestion for the past nine months, but she had never had any hæmatemesis, nor indeed any of the more acute symptoms associated with gastric ulcer. On the morning that the perforation took place she felt quite well and had taken her breakfast as usual, which consisted of a plate of porridge and two cups of tea. Immediately after breakfast, and while sitting at the table, she was seized with very severe pain in the epigastrium and felt 'that something had given way inside.' On trying to walk to the door she fell to the ground, vomited, and became collapsed. Dr Ballantyne saw her about two hours afterwards, and diagnosed a perforated gastric ulcer. When I saw her two hours later, she was still suffering from collapse, and complained of severe pain all over the abdomen; the abdominal muscles were tense, but there was little or no diminution in the area of the liver dulness. She had vomited several times, and her fæces had passed involuntarily. The pulse was small and rapid. As there seemed to be no doubt about the correctness of Dr Ballantyne's diagnosis, I proceeded at once to operate. The abdomen was opened in the middle line above the umbilicus. Nothing abnormal was discovered upon the anterior wall of the stomach, but on passing the finger through the foramen of Winslow, there issued an ounce or two of dirty-looking serous fluid, mixed with small flakes of lymph. On opening into the lesser sac of the peritoneum, so as to expose the posterior wall of the stomach, the latter was

found to be intensely congested and dotted here and there with flakes of lymph. It was some time, however, before the perforation could be discovered. It was observed that the lymph became more abundant as one passed towards the cardiac end of the stomach. Ultimately, a perforation about three or four millimetres in diameter was found just below the lesser curvature, close to the œsophageal opening. It was with difficulty that the perforation could be brought into a position accessible for suturing; with considerable traction, however, this was achieved, and the opening closed by a double row of sutures. The abdomen was then irrigated with a large quantity of warm sterile water. A drainage tube having been introduced into the lesser sac, the rest of the abdominal wound was closed by silk-worm gut sutures passed through the whole thickness of the abdominal wall. The tube was removed on the tenth day, the stitches on the twelfth day. The patient made an uninterrupted recovery, and she has since been almost free from indigestion."

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The points of interest in this case are:—(1) The early diagnosis and the early operative treatment; (2) the presence of a normal area of liver dulness; (3) the healthy appearance of the abdomen until the lesser sac was investigated; (4) the difficulty in reaching the perforation, in consequence of its position on the posterior wall close to the œsophagus. In relation to the position of the ulcer, it is interesting to note that for several weeks before perforation the patient, immediately after the act of swallowing, complained of a raw sensation behind the lower end of the sternum.

(*b*) Complete AVULSION OF THE SCALP, showing result of skin-grafting.

Mr Stiles reminded the society that he had exhibited this patient to the members of the society about a year ago, when the pericranium was covered with very vascular and hypertrophied granulations. While skipping in a public laundry, first the rope, and then the patient's hair, was caught by a revolving wheel, and the entire scalp stripped off. The first attempt at Thiersch-grafting had failed. The patient at that time was in such a weak state of health that it was not deemed advisable to remove the granulations before applying the graft. The result was that although the grafts adhered and became vascularised, they were ultimately eaten up by the granulations.

The subsequent grafts were applied to areas from which the granulations had been removed, the hæmorrhage, which was considerable, being checked by a solution of peroxide of hydrogen. After trying various dressings, the house surgeons (Drs Chiene and M'Intosh) who did the grafting, came to the conclusion that the most satisfactory dressing consisted of strips of perforated protective covered with dry sterilised gauze smeared with a weak antiseptic ointment. The whole scalp is now covered with sound epidermis, except at one or two points where the cicatrix is weak and slightly excoriated. Fortunately the patient has a slight fringe of hair in the frontal and occipital regions, so that with her hat on her deficiency is not noticed.

(*c*) An infant, three months after removal of a large OCCIPITAL MENINGOCELE, 16½ inches in circumference.

The infant was admitted to the Children's Hospital when it was three months old. Springing from the occipital region and attached to it by a pedicle 5½ inches in circumference, was a globular translucent tumour which had the feel of a tensely filled cyst. It did not become larger or tenser when the child cried. The skin over it was here and there excoriated and eczematous. The cutaneous veins were very much dilated, especially towards the pedicle. The infant was slightly hydrocephalic; the head was elongated anteroposteriorly, the forehead prominent, the vault high, and both fontanelles were enlarged. In removing the cyst, two flaps of scalp were dissected off the membranes down to an opening, the size of a sixpence, in the occipital bone. A stout catgut ligature was then tied round the pedicle, and the latter divided. Projecting from, and included in, the pedicle, was a clump of arachnopia, which was ligatured separately and the redundant portion removed. No cerebro-spinal fluid escaped. The two short flaps of scalp were then brought together over the pedicle and sutured in such a way that their surfaces as well as their edges were brought into contact. The patient left the hospital on the thirteenth day. The infant is now distinctly more hydrocephalic than at the time of operation. The opening in the occipital bone can be distinctly felt, and there is a slight tendency of the membranes to herniate through it.

(*d*) An infant, aged four months, after operation for ACUTE INTUSSUSCEPTION.

The patient was sent into the Children's Hospital by Dr

Kirk, of Bathgate. The operation was performed twenty-two hours after the intussusception occurred. The history was very typical. At six o'clock the previous evening, the infant, while at the breast, took a violent attack of screaming, and appeared to be in great distress. Attacks of spasmodic pain and screaming occurred at intervals throughout the night. The following morning the mother noticed the escape of blood and mucus per anum. Vomiting next set in, and occurred several times. On admission to hospital, at four o'clock the same afternoon, the infant was found to be somewhat collapsed. The configuration of the abdomen was normal, but a firm and somewhat sausage-shaped swelling could be felt in the left lumbar region. The right iliac region felt abnormally empty. The abdomen was opened in the middle line by a three-inch incision, the centre of which skirted the umbilicus. The intussusception which was deeply placed in the left lumbar and left hypochondriac regions, could not be brought into the wound until the greater part of the intestines were brought out of the abdomen. There was no peritonitis. The distal portion of the intussusception could be reduced with ease by manipulation through the sheath; as usual, however, the last, or ileo-cæcal, portion was more difficult to reduce, in consequence of the œdema and swelling, which is always more marked at this region. No adhesions had formed between the entering and reflected layers of the intussusceptum, and the bowel, although somewhat œdematous, was otherwise healthy. The wound in the abdominal wall was closed by silk-worm gut sutures passing through its whole thickness. The operation did not take more than a quarter of an hour, and in a few hours the child had recovered from the shock. During the next three days the motions were distinctly tarry. The child made an uninterrupted recovery and left the hospital on the eighteenth day.

Mr Stiles pointed out the importance of early diagnosis and early operation in cases of acute intussusception; he believed that if the abdomen be opened within twenty-four hours after the occurrence of the invagination that fifty per cent. of the cases ought to recover. He did not, therefore, take the gloomy view which is almost universally held regarding the prognosis in acute intussusception.

(*e*) Boy with PARALYSIS OF GLUTENS MAXIMUS, the result of acute anterior poliomyelitis.

The patient was brought to the Out-patient Department of the Children's Hospital on account of lameness. On stripping him and making him walk across the room it was at once noticed that his gait was peculiar, and not at all like that seen in disease of the hip, knee or spine, and yet there was distinct atrophy of the gluteal region, and to a very slight extent also of the thigh. There was no apparent or real shortening of the limb, and he walked with the whole sole planted firmly on the ground. Further examination showed that the movements at the hip were quite free and of full range. The spine was normal, and there was nothing abnormal to be discovered either in the iliac fossa or in the groin. He had never complained of pain. His gait was not unlike that which we sometimes see in boys with phimosis and preputial adhesions, and which is the result of a slight reflex paresis and want of co-ordination; but there were no preputial adhesions. On examining the buttock more carefully, it was found that the glutens maximus was flabby and atrophied, and could not be thrown into contraction either voluntarily or by the galvanic or faradic current. The glutens medius and minimus were normal. The boy's mother volunteered the interesting information that he seemed to be most lame while going down a stair. This, which was found to be the case, was evidently due to the loss of the extensor action of the glutens maximus. When he walked upon the level it was observed that it was the imperfection of the backward movement at the hip which created the lameness. Mr Stiles thought the case was of interest because of the rarity of the condition; moreover, it illustrates the importance of keeping before one the possibility of such rare lesions when searching for a cause in the more obscure forms of lameness, which are sometimes met with in children.

7. *Professor Annandale* and *Dr Alex. Bruce* exhibited a patient after LAMINECTOMY FOR PARALYSIS due to Pressure on Nerve Roots.

The earlier history of the patient, who is an upholsterer, aged 28, was published by Dr Bruce in the form of a clinical lecture in the February number of the *Edinburgh Medical and Surgical Journal* of 1900. "A case of traumatism of part of the cauda equina, complicated by alcoholic neuritis." It was briefly as follows:—A fall downstairs followed by complete

paralysis of the left lower extremity with an area of anæsthesia corresponding to the distribution of the twelfth thoracic or dorsal, the five lumbar and the first sacral nerves. To this condition there was superadded later, paralysis of the right leg and foot, hyperæsthesia to pressure upon the muscles of the right lower extremity, and of the muscles underlying the still æsthetic areas of the skin of the left lower limb.

Reasons were given for the view that the paralysis was due to some form of pressure by the lamina of the twelfth dorsal vertebra, as that was the only structure which could affect *all* these roots without any further symptoms. The patient was sent to the North of Scotland to his home, with instructions to remain a total abstainer from alcohol and to return for operation so soon as he had recovered power in the right foot. This he accordingly did in the month of July 1900, and after a short stay in the medical ward under Dr Bruce, he was transferred to Professor Annandale's care.

Professor Annandale reports as follows:—"In July last my colleague Dr Bruce consulted me in regard to this patient. He told me that the man had received a direct injury to his spine, and from a study of his symptoms he was of opinion that there was some pressure or irritation upon the spinal roots at a point corresponding to the twelfth dorsal vertebra. Having by examination confirmed Dr Bruce's diagnosis, he and I agreed that an exploration of this region was advisable. Accordingly on the 15th July, the patient having been chloroformed, I explored the transverse processes and laminæ of the tenth, eleventh, and twelfth dorsal, and first and second lumbar vertebræ, by turning up a flap of the soft textures from left to right. When the bones were exposed nothing abnormal was noticed, but when the spinous processes and laminæ of the twelfth dorsal and first and second lumbar vertebræ were removed, and the spinal canal thus freely exposed, there was seen to be a marked contraction of the spinal cord at a point corresponding to the twelfth vertebra, this contraction being due to a distinct narrowing of the spinal canal. This narrowing of the canal and contraction of the cord was limited to an extent of about half an inch. No other abnormal condition of the cord or its membranes could be detected. The wound having been closed by sutures, superficial and deep, a drain of iodoform gauze was inserted. His progress after the

operation was in every way satisfactory, and on the day following the operation it was noted that the motor power and sensibility of the left foot had already improved. This improvement slowly continued, the wound healed well, and he was transferred to Dr Bruce's medical ward upon 1st September."

The patient remained in the medical house and in the Convalescent Hospital under the care, successively, of Dr Bruce and Professor Fraser. During that time there was a steady, if slow, improvement of motor power in the lower limb, but only a very slight diminution of the anæsthesia.

The slow improvement of the lower extremity was accounted for by the fact that the muscles had undergone atrophy to a large extent during the long time which elapsed between the original injury in February 1899 and the operation in July 1900. This atrophy was indicated by the completeness of the reaction of degeneration present in all the muscles. The recovery was most complete and earliest in the muscles of the hip, least complete in the foot. It is not necessary to enter into the details of the progress of the case. At the time of his leaving the hospital the resident physician, Dr Arthur Preston, reported that at the hip-joint the limb could be abducted, adducted, crossed over the other limb, advanced and retracted almost to the normal amount, and rotated outwards and inwards to a more limited extent. He could bend the knee only very slightly, even when lying in bed. The patient could not dorsiflex the ankle, but if it is passively dorsiflexed he could extend it again. He could flex all the toes except the great toe. He required crutches in walking, swinging his right leg forward from the hip-joint. He was able to bear a considerable amount of his weight on the right leg. The anæsthesia had diminished to a very slight extent, practically only over the area corresponding to the supply of the first sacral root.

Since leaving the hospital the patient has written to the effect that the strength of the limb has considerably increased, but that the movements of the foot and ankle are still imperfect.

In considering the causes of the slow return of motor power and imperfect recovery from the anæsthesia, it has to be remembered that the long continued pressure on the nerve

roots had led to a degeneration of the nerves and of the muscles of the lower limb, and that recovery of motor power had to be preceded by a regeneration of both nerve and muscle fibres. The fact that the recovery of motor power was greater in extent than that of sensation is probably to be attributed to the circumstance that the pressure of the affected lamina must have been exerted to a greater extent on the posterior than on the anterior roots, and had caused damage of a permanent nature to these roots. The cause of pressure was evidently due to a thickening of the lamina from chronic periosteal irritation, whether the result or not of a fracture it was not possible to determine definitely. The examination of the lamina gave no indication of any such injury.

II. Exhibition of Pathological Specimens

1. *Dr Allan Jamieson* exhibited microscopic specimens from case of CERVICAL TUMOURS shown at the November meeting.

The growths were completely excised by Professor Annandale, and sections were prepared by Dr Welsh. At the meeting the opinion was hazarded that the condition was epitheliomatous or carcinomatous, but this was not borne out by the examination. The scar-like tissue and the elevated nodules exhibited practically the same structure. The epidermic layers were thinner than normal, and the epithelial pegs flattened out, but there was no morbid alteration of the cells, and no dilatation of lymph spaces. Dr Welsh says "the scar tissue shows great atrophy of the skin structures, and a dense over-growth of fibrous tissue. The smaller nodules are mostly close beneath the skin, and have a looser and more cellular structure of a myxomatous type. The larger nodules are also close beneath the skin, are very cellular with many immature blood-vessels, and rather resemble a spindle-celled sarcoma." To this may be added that the coil glands are natural in appearance, there is some cell infiltration in the neighbourhood of the hair follicles, and the sebaceous glands in one specimen showed an alteration, which seemed of a colloid nature, in certain of their cells.

2. *Mr Alexis Thomson* exhibited the DEFORMED SHAFT OF THE TIBIA, removed from the patient exhibited at this meeting.

The portion of tibia removed exhibited on section two distinct circumscribed abscesses, each about the size of a plum, lined by a delicate layer of granulation tissue, and filled with greenish yellow pus. The bone surrounding the abscesses, and throughout the shaft generally, was sclerosed; the medullary canal was obliterated.

3. *Mr J. Shaw M'Laren* exhibited—

(*a*) A recent POTT'S (?) FRACTURE. Fibula broken transversely much lower down than the site usually described in Pott's fracture. Also a transverse fracture of the internal malleolus. If it was not a Pott's fracture, the *symptoms* were certainly typical.

Radiograph of FRACTURED INTERNAL CONDYLE OF HUMERUS lodged in the sigmoid cavity, between the humerus and ulna. The joint could not be flexed beyond a right angle. The joint was accordingly opened, the fragment, attached to some of the flexors, withdrawn, and nailed in its proper place.

(*b*) Radiograph of FRACTURE OF THE ASTRAGALUS with subastragaloid dislocation of the foot forwards. Knowledge gained by the Röntgen rays favours the idea that perhaps fractures of the astragalus are not so rare as was formerly supposed. This forward dislocation is much rarer than the backward.

4. *Mr H. J. Stiles* exhibited—

(*a*) Portion (3½ inches) of HATPIN REMOVED FROM MALE BLADDER by suprapubic cystotomy.

The patient, a healthy middle-aged bachelor, had introduced the pin, head first, into the bladder. He stated that his object in introducing the pin was to investigate the cause, and if possible also to bring about a cure, of the seminal emissions about which he had become anxious. Unfortunately the point of the pin disappeared within the urethra and the patient found himself in a dilemma. Dr Stirling, of Perth, who was called in, removed the anterior half of the pin through an incision made into the spongy portion of the urethra a little in front of the bulb. When I saw the patient on the following morning I passed a sound and felt the pin, which appeared to be fixed in the bladder. Assisted by Dr Stirling, I opened the bladder suprapubically and removed the pin, which was

lying in the transverse axis of the bladder and rather firmly gripped by it. The patient's recovery was somewhat retarded by some deep suppuration in the cellular tissue to the right of the bladder in the region where the latter had probably been wounded or perforated by the pin. The specimen is rather a "rara avis" as a foreign body in the male bladder.

(*b*) BRAIN SHOWING ABSCESSES secondary to an injury to the head.

Nine weeks before admission to hospital the child (aged 3 years) was struck on the back part of the left frontal region by a poker which the father had thrown at the mother, who had the child in her arms at the time. For two or three days after the injury the child was drowsy, vomited frequently, and exhibited twitchings of the opposite arm and leg. These symptoms disappeared in a week, and the child seemed practically well, except that the wound suppurated and took eight weeks to heal. A few days after it had apparently healed a pulsating swelling, about 1 inch in diameter, developed at the anterior end of the cicatrix. Three days before admission vomiting again set in, and the child began to get listless. On the day after admission to hospital a flap of scalp, including the scar, was dissected down off the pulsating swelling, and it was seen that the child had sustained a comminuted fracture. The fragments of bone were adherent but somewhat eroded, and there were unhealthy looking granulations and some strands of hair in contact with the dura. After removing some of the adherent pieces of bone and turning down the dura, a small cortical abscess was discovered. The parts were then cleaned up and the flap of scalp secured in position by two or three stitches, leaving space between them for drainage. Next day the child was much brighter and wished to sit up: there was no paralysis. A day or two later the flap bulged considerably, and a hernia cerebri developed and enlarged rapidly. The patient was again chloroformed and the hernia investigated, but no accumulation of pus could be discovered in connection with it. The hernia, however, continued to enlarge, the right leg and arm became paralysed, drowsiness and vomiting set in, and the child died eighteen days after the first operation. At the sectio two abscesses, both about the size of a walnut, were found in the left occipital lobe. The one abscess had set up a

suppurative basal meningitis, while the other had encroached upon the lateral venticle, which contained about a teaspoonful of pus.

The case is a good illustration of the fact that when a rapidly enlarging hernia cerebri follows a cerebral injury, suppurative encephalitis is generally the cause.

(*c*) CONGENITAL FATTY TUMOUR from axilla of an infant aged 3 months.

The tumour, which was noticed soon after birth, had grown rapidly during the past six weeks. It measured 4 inches in diameter, was distinctly lobulated and well circumscribed, though not very prominent; it had a characteristic doughy consistence, which gave the feeling of a false fluctuation. The veins of the skin were not dilated.

The diagnosis lay between cystic lymphangioma, nævo-lipoma, and simple fatty tumour. On removal the growth proved to be a soft encapulated and lobulated fatty tumour, the deep surface of which was adherent to the serratus magnus.

In infants such tumours are by no means common.

5. *Dr Harvey Littlejohn* exhibited—

(*a*) BRAIN SHOWING CEREBRAL HÆMORRHAGE, death by suicide six weeks afterwards.

The man was 60 years of age. He was suddenly seized with partial paralysis of the left side, together with interference with his sight, hearing, and speech. During the following four weeks he gradually improved, so much so, that he thought he was able to resume his work as a zincographer. This employment, however, requires good eyesight, and he found that he was unable, on account of some defect in accommodation, to perform his duties. The fact depressed him very much, and he was found one morning with his throat cut. He left home after breakfast perfectly cheery and well, and gave no indication of an intention to take his life.

(*b*) CUT THROAT, from the above case.

This presented a peculiarity in as much as the incision which ran from behind the left ear to 2 inches beyond the middle line of the neck on the right side, passed above the hyoid bone, and completely divided various muscles attached to it. The incision did not, however, enter the pharynx.

The external jugular vein on the left side was completely divided, and the external carotid artery was cut into.

(*c*) A SPECIMEN SHOWING COMPLETE TRANSVERSE RUPTURE OF THE SMALL INTESTINE, also RUPTURE OF THE MESENTERY, the result of a simple fall.

A man, somewhat the worse of drink, was proceeding home on a Saturday night along the railway, and, according to his own account, he stumbled and fell forwards across one of the rails. He picked himself up and went to his house, and although complaining of pain in the lower part of the abdomen his condition did not rouse special attention, and he apparently slept quietly all night. Next morning he was evidently ill, complained of pain, vomiting commenced, and a doctor was sent for, who, upon arrival in the afternoon of Sunday, ordered his removal to an hospital. This hospital was eight miles distant, and the patient died before arriving there.

At the post-mortem examination there were no signs of external injury, but on opening the abdomen a loop of small intestine was found completely divided across; while there was also a rent running down for 2 inches through the mesentery.

The interest in the case lies in the fact of such a severe rupture being occasioned by a simple fall.

(*d*) A SPECIMEN OF DOUBLE UTERUS.

This specimen was removed from a woman in whom the condition was unknown to exist, and who had borne a child eight years ago after a normal labour. The uterus was completely double, and a well-marked septum divided the cervix. The vagina had also evidently been divided as there were two distinct fleshy ridges, half an inch in depth, on the anterior and posterior vaginal walls.

(*e*) A STOMACH SHOWING ACUTE GASTRITIS.

A woman, about 40 years of age, developed a slight erysipelas on the face. She was removed to an hospital, and died two days afterwards. During this period there was a considerable amount of hæmorrhage from the bowel, the blood being red and unchanged. She also vomited coffee ground material on two or three occasions.

The erysipelas remained slight, and circumscribed, and she apparently died from exhaustion, in consequence of the loss of blood. At the post-mortem the various organs were found to

present a comparatively healthy and normal appearance, with the exception of the stomach and the upper part of the small intestine, which, externally, were dark red and inflamed in appearance, although there was no sign of peritonitis.

The mucous membrane of the stomach and upper part of intestine was swollen, velvety, and of a uniform dark red colour. This was due to hyperæmia, and to extravasation of blood in the sub-mucous tissues.

No corrosion or other lesion could be detected in any part of the mucous membrane of the stomach, or in the small and large intestines and rectum, and although a careful and minute examination was made, the source of the hæmorrhage was not discovered. It must apparently have been due to a general transudation of blood through the mucous membrane.

Meeting IV.—January 16th, 1901

Mr A. GORDON MILLER, *President, in the Chair*

I. ELECTION OF MEMBERS

The following were elected Ordinary Members of the Society:—J. E. Moorhouse, M.D., C.M., Stirling; David Lawson, M.A., M.D., Banchory; D. A. Carruthers, M.B., C.M., Perth; and J. M. Rutherford, M.B., C.M., Royal Asylum, Edinburgh.

II. EXHIBITION OF PATIENTS

Mr F. M. Caird exhibited four cases of PYLORIC OBSTRUCTION treated by gastro-jejunostomy—

1. D. M., æt. 35, an alcoholic, suffered from symptoms of gastric ulcer in 1896, but enjoyed good health thereafter till 1899, when pain and vomiting started. Operation, posterior gastro-jejunostomy, 31st December 1900. Hard tumours and many adhesions on the pylorus. Many enlarged lymphatic glands. Stitches removed, and healed 16th January 1901. Patient steadily gains weight.

2. T. M., æt. 44. Large mobile carcinoma of pylorus, with glands involved. Symptoms date from August 1899. Anterior gastro-jejunostomy, December 22, 1900, when he

weighed 8 st. 10 lb. Healed about 16th January; weight, 8 st. 13 lb.

3. A. T., æt. 48. Very large mobile tumours; no glands. Symptoms date from 1897, when he weighed 11 st. Operation of anterior gastro-jejunostomy, 26th December. Weighed 8th January, 6 st. 6½ lb. Healed, and weighed 6 st. 11½ lb., 16th January 1901.

4. Mr L., æt, 68. Symptoms date from spring, 1899. Large mobile tumour and glands. Weight, 7 st. 2 lb. Anterior gastro-jejunostomy, 14th December 1900. Healed, and weighed 7 st. 5¾ lbs., 8th January 1901. Taking ordinary diet with avidity, and weighed 7 st. 7 lb. 16th January 1901.

III. DEMONSTRATIONS

1. *Dr Chalmers Watson* gave a demonstration on the comparative PATHOLOGY OF ATAXIA. The animal which afforded the specimens was a young adult cart-horse, 9 years old, which presented symptoms of ataxia, slight paraplegia, and a bilateral affection of the joints of an unusual nature. The animal could walk, trot, and even gallop for a short distance, but all of these movements showed indications of the weakness referred to. The ataxia was most marked when the animal was turned round and also when an attempt was made to back it. This latter movement was impossible, and the attempts showed that the hind feet were markedly misdirected. The combination of symptoms led to the diagnosis of a lesion of the spinal cord, involving mainly the posterior columns in the lumbar region, with an associated neurotrophic lesion of the knee joints. This was verified at the post-mortem examination. The demonstration included a series of specimens from the lumbar and dorsal cord; photographs of the knee joints, showing very extensive disease, involving mainly the soft parts; and a number of large addimentary bones removed from the neighbourhood of the knee joint. The special object of the histological demonstration was to show that vascular disease in the cord was the primary lesion, the involvment of the neurons being a secondary occurrence. The case, which is believed to be unique, will be fully recorded later.

The findings in this case, in addition to shedding some light on the hitherto unknown etiology and pathology of some

diseases of the horse, raises some very interesting questions as to the correctness of the views at present held regarding the etiology of allied diseases in the human subject.

2. *Dr Dawson Turner* gave a demonstration on an IMPROVED RÖNTGEN APPARATUS.

It has lately been recognised that the nature of the interrupter of the coil used to obtain Röntgen-rays is of extreme importance. I desire to show the Medico-Chirurgical Society to-night three of these instruments.

(*a*) Mackenzie Davidson's mercury interrupter.

(*b*) A simple mechanical interrupter of my own construction.

(*c*) Wehnelt's electrolytic interrupter.

The last gives by far the most powerful discharges. It requires, however, an electro motive force of at least 30 volts, as it is apt to tire and to overheat the tubes.

Instantaneous skiagraphs can, however, of comparatively thick parts, be obtained, and these have the advantage of giving greater sharpness of detail, and of sometimes exhibiting the soft parts. I show some instantaneous photographs of the knee and elbow.

IV. ORIGINAL COMMUNICATION

THE EDINBURGH HOSPITAL IN SOUTH AFRICA AND ITS WORK

By FRANCIS D. BOYD, M.D., F.R.C.P.Ed., Physician to the Edinburgh Hospital in South Africa, Assistant Physician to the Royal Infirmary, Edinburgh, etc., and GEORGE L. CHIENE, M.B., C.M., Assistant Surgeon to the Edinburgh Hospital in South Africa

THE account of the Edinburgh Hospital was opened with a lantern demonstration by Dr Boyd of views of the hospital from the earliest days at Norval's Pont to the time of completion. The building of the hospital, its working, sanitation, water supply, and other points, were in turn dealt with. Dr James Miller next gave an account of the 57 cases dealt with in the X-Ray department. Dr Chiene gave an account of the surgical work of the hospital, and thereafter Dr Boyd read his paper on the medical work of the hospital.

The Surgical Work of the Hospital

By Mr GEORGE L. CHIENE

Mr President and Gentlemen,—Dr Boyd, with the permission of the Society, has asked me to show some lantern slides demonstrating the various forms of bullet wounds treated in the Edinburgh and East of Scotland South African Hospital, and to say a word or two about the surgical part of the work of the hospital.

I am very sorry that Mr Wallace, owing to his unfortunate illness, was unable to superintend the surgical work. Dr Watt was to have taken his place to-night, but he has within the last week gone back to South Africa.

To Dr James Miller we are indebted for the X-ray photographs and for the notes regarding them.

The total number of surgical cases treated was 166 (this does not include out-patients, a great number of which were surgical).

The surgical cases treated in the hospital, with the exception of a few, were all admitted on or after July 26th, the chief work of the hospital up to that time being of a medical nature.

Of the above cases 75 were bullet or shell wounds—65 due to bullet, 10 due to shell wounds.

The great majority of the above were wounds of the extremities, as is seen from the following table:—

Head and neck	8
Thorax	10
Abdomen	8
Extremities	49

The other cases, which we may combine under the head of general surgery, included injuries, burns, fractures, abdominal cases; tubercular, urinary and specific disease.

We had three deaths, one a compound fracture of the base of the skull and compound fracture of both legs. Two were moribund abdominal cases, one of which was a perforation due to typhoid.

The cases of general surgery are of no special interest to us at present, except from the fact that a great number of them were directly due to the campaign.

I will now show one or two photographs of cases of general surgery on the screen, and then some demonstrating the varieties of bullet wounds treated by us at Norval's Pont. Before doing so, however, I would draw attention to the bullets and pathological specimens on the table.

Photographs of the following cases were then shown :—

1. A marked case of varicose veins requiring operative interference.

2. A case of acute middle ear disease following typhoid fever, also operated on.

3. A case of gangrene of both feet following an attack of pneumonia. The one leg was amputated at the seat of election, and on the other foot a Syme's amputation was performed.

X-ray photographs, illustrating five cases of shrapnel wounds, followed. All, with the exception of one, were septic on admission.

The first case was one of a severe compound fracture of the radius, the photograph showing the splintering of the bone.

The photograph of the second case showed a compound fracture of the humerus with the shrapnel *in situ*. The septic cavity was opened up and the bullet removed.

The third showed a shrapnel bullet embedded in the outer condyle of the femur. The bullet was removed.

The fourth showed splinters of shrapnel in the region of the wrist joint.

The only aseptic case, of those shown, was the last: a shrapnel bullet lying in the upper part of the adductor longus. The bullet was removed, and the wound healed in a week.

X-ray photographs illustrating five cases of bullet wounds were then shown : four due to Mauser bullets and one caused by a Lee-Metford. All were aseptic on admission except the first, which was a severe compound fracture of the metacarpal bones of the hand requiring operation.

The second showed a bullet in the region of the spine on a level with the iliac crest. The case was operated on, but the bullet was not found. The wound healed without trouble.

The third case was one in which the bullet was embedded in the head of the tibia. The knee joint was opened and the bullet removed. It was found to be a Lee-Metford. The patient recovered with a stiff knee.

The fourth case was one of special interest. The bullet was seen lying in the head of the left fibula, having first passed through the right thigh. Some fragments of the bullet could be seen lying in the region of the popliteal artery of the left leg. The bullet was removed from the head of the fibula, no attempt being made to remove the fragments. A short time afterwards a popliteal aneurism developed. It was found necessary, at a later date, to ligature the femoral artery at the apex of Scarpa's triangle.

The photograph of the fifth case showed a bullet lying on the anterior surface of the femur. The bullet was removed through an incision on the outer aspect of the thigh.

Several photographs were then shown of a soldier whose lower jaw had been carried away by a pom-pom. The operation was done in three stages. The patient was then shown along with his photograph which was taken previous to his departure for South Africa.

The Medical Work of the Hospital

By DR BOYD

DURING our time at Norval's Pont, we admitted between five and six hundred patients and prescribed for eighteen hundred out-patients. Of the medical cases, the principal disease dealt with was enteric fever. We admitted in all, a little over 150 cases of the disease. The onset of the disease was in almost every case identical—pain in the back of the head and in the neck and limbs. The bowels were, as a rule, constipated, diarrhœa was the exception.

The marked characteristic was the extreme foulness of the mouth accompanied by inflammation of the fauces. In many of the cases this might be ascribed to unavoidable neglect, but it was present even in the cases amongst the staff when the greatest precautions were taken from the outset of the disease. Ulceration of the soft palate, the so-called pharyngo typhoid, was only seen once. Melæna occurred in only two cases; in one it was slight, in one it proved fatal in a few hours.

Considering the frequency of dust storms, it is rather sur-

prising to find how few of the cases exhibited affections of the upper respiratory tract. In one there was laryngitis with aphonia. In one œdema of the glottis, which necessitated tracheotomy. The patient, much to our disappointment, died nineteen days after operation, from asthenia and bronchitis, the tube having been removed two weeks before death.

Hypostatic congestion of the lungs was met in 15 per cent. of the cases. No case of true crupous pneumonia occurred as a complication, though broncho-pneumonia was fairly frequent. Infarction of the lung with hæmoptysis was seen in two cases. Pleurisy with effusion in four cases. Hiccough, a somewhat rare complication, except in connection with diaphragmatic peritonitis, was in one case a very serious feature. The patient hiccoughed for nine days; for three days the hiccough was continuous night and day, entirely preventing sleep, and gave rise to grave anxiety. There was no peritonitis, and the patient made a good recovery. In three cases old standing cardiac lesion was discovered on admission. Of these three cases two died, demonstrating what a serious handicap an old-standing cardiac lesion is in enteric.

Thrombosis was present as a common complication, occurring in 6 per cent. of the cases. It came on late in the disease, occurring in the earliest case on the *twenty-sixth* day. There was, as a rule, a rise of temperature, with pain along the course of the veins, usually the veins of the leg, and œdema of the limb. In one case thrombosis of the branches of the middle cerebral artery gave rise to paralysis with aphasia. One is at a loss to account for the frequency of thrombosis as a complication. It is rare in America, though frequent enough in Great Britain. It has been ascribed to putties, but our cases had been, in the earliest case, twenty-six days without putties being worn, and putties should, if properly applied, have a supporting, not an injurious influence on the veins. Nephritis and cystitis were met but rarely, and they only occurred in patients who started with a damaged urinary tract.

Relapse occurred in 5·3 per cent. of the cases, a low proportion when dealing with male patients alone. There was no mortality from relapse. Chiari has suggested that the cause of relapse is a storing up of bacilli in the bile ducts. On any error in feeding, extra strain being thrown on the liver, the

bacilli are discharged into the alimentary canal and a fresh infection takes place. Our experience does not agree with this. There was no error in feeding in most of the cases, and in addition in the fatal cases, as can be seen from Dr Miller's specimens, no bacilli were found in the liver or bile ducts. There is, however, marked reactionary changes and stasis in the mesenteric glands. It seems more probable that reinfection takes place from the mesenteric glands on subsidence of the reaction in the glands, the poison being set free as the lymph stream is re-established.

The fever, as seen in South Africa, may be divided into three types: the mild or abortive, the grave, and the apyrexial. Of the mild type a number of cases were seen. The patient had headache, malaise and gastro-intestinal disturbance, with a mild fever lasting six to eight or ten days. Of the grave type there were two distinct classes. In the one the gastro-intestinal symptoms predominated. There was a very foul tongue, the abdomen was markedly distended, nourishment was poorly taken and badly assimilated. If the nourishment was pushed, the stools showed the presence of undigested milk. If the case proved fatal, very extensive ulceration of the small intestine was found. In the second variety, which might be described as the toxæmic, the rash was marked, the temperature high, and the nervous symptoms predominated, while the abdominal symptoms were subsidiary. Only one case of the apyrexial type of the disease was noted, in one of the members of the staff who had been inoculated. He had a rash, gastro-intestinal symptoms, enlarged spleen, and typical stools, but the temperature during the five weeks he was in hospital only once rose to 99·4; as a rule it was subnormal. The condition appeared to be a mild attack of enteric modified by inoculation.

Widal's reaction was carried out in all doubtful cases, and in most cases where the diagnosis was certain. The dilutory medium used was either bouillon or normal saline in a dilution of 1 in 30. There were always three to six observations going on, so a check was thus established. The culture used was 24 to 36 hours old.

An account was then given of nine cases of apparent enteric fever where the agglutinative test proved negative, and the diagnosis discussed.

"Simple Continued Fever"

In the Army classification of disease "simple continued fever" is to be found as a distinct entity. It is probable that it owes its presence to the fact that the authorities desire a definite diagnosis for weekly returns, and it may at the time be impossible to say what febrile condition the patient is developing. After a few days the condition declares itself to be possible enteric fever. The diagnosis is not changed in the returns—it cannot be. The patient must be nominally discharged as simple continued fever, and readmitted as enteric fever. But is there such a disease as "simple continued fever"? Most army medical men who have had large experience in tropical and sub-tropical climates assure me that there is a definite febricula which occurs in white people in hot climates who have undergone fatigue and been exposed to the influence of a tropical sun. The civil surgeons, on the other hand, were for the most part inclined to regard all cases of "simple continued fever" as undiagnosed enteric fever.

It is interesting to consider the cases which were admitted to the Edinburgh Hospital, for we had the means, by examination of the blood, of more or less certainly excluding enteric fever.

In all, we admitted 38 cases. In ten of these no definite diagnosis could be arrived at. The patient was seedy and run down. There was no fever while in hospital. He had a history of headache, and possibly a day of doubtful fever. When under our care there were no physical signs of disease; the Widal's reaction was negative. It is possible, I think indeed probable, that those patients were for the most part convalescents after influenza. There was a considerable amount of influenza in South Africa at the time, and I am inclined to think that influenza will account for most, if not all, of the ten cases. Of the remaining 28, seven were undoubtedly cases of enteric fever of a mild or abortive character. There was the usual history of headache, malaise, constipation, and some fever lasting a few days. Examination of the blood gave a positive Widal's reaction. Four cases may have been abortive enteric fever. There was a similar history of malaise, fever for a few days, etc., and the examination of the blood was positive, but then the patient had been inoculated against enteric so no

diagnostic evidence could be drawn from the Widal's reaction. In six cases there was a history of former repeated attacks of malaria fever. The patient when he came to us had as a rule been dosed with quinine; thus in none of the cases were we able to prove the presence of the malaria parasite, but the fever must be considered to have been of malarial origin. Take a soldier who has had repeated attacks of malaria, expose him to the influence of a hot sun by day, cold by night, couple this with fatigue and possibly damp. Such a man being predisposed through his malaria to febrile reaction, I see no reason to doubt that under the circumstances a febrile attack will be precipitated. The same applies to Mediterranean fever. Two of the patients admitted had had repeated attacks of Mediterranean fever. When under our care they passed through a mild febricula which was certainly not enteric fever, but no more was it a definite attack of Mediterranean fever. Of the other cases of "simple continued fever," two were catarrhal dysentery; one, debility from overmarching, with some slight dilatation of the right side of the heart; convalescent pneumonia accounted for one; rheumatism for one; gastro-intestinal catarrh for one; one was a surgical case; one the results of salivation with mercury; and one an early pulmonary tuberculosis. From these facts one may conclude that "simple continued fever," while it is a useful term for a preliminary diagnosis, can scarcely be elevated into the position of a distinct disease. As a rule, if the case is watched with care, a definite diagnosis can be arrived at. The figures are of interest from another point of view: if 18 per cent. of the cases of "simple continued fever" are enteric fever, it is very desirable to have more definite diagnosis, or else sanitary precautions should be taken in all febrile conditions where enteric cannot be definitely excluded.

Anti-Typhoid Inoculation

Since the occurrence of the disastrous epidemic of enteric fever in South Africa, medical men, and one might, with justice, say the public in general, have looked forward with great interest to an account of the effects of inoculation on the disease. Has inoculation proved as valuable as was hoped, not only in preventing, but in modifying the disease? I fear

that it is impossible from the experience of a single hospital to give a satisfactory answer to such a question. We must wait till the statistics of the whole campaign are published before we can draw any definite conclusion, and even then these statistics will require very careful consideration.

Inoculation is based upon the fact that when animals are inoculated with dead cultures of typhoid bacilli, an increased power of resistance against the invasion of the living typhoid bacillus is established. In man, it has been proved that the injection of an emulsion of dead typhoid bacilli produces the same blood changes that a similar injection produces in animals. It has further been shown that the same blood changes are produced in man by the injection of an emulsion of dead typhoid bacilli as are produced by an attack of enteric fever.

Of the staff of the Edinburgh Hospital 58 were inoculated; 27 being done once, and 31 twice. In 7 the inoculation appeared to produce no appreciable effect. In a smaller number there was merely a slight local reaction as indicated by some tenderness at the point of inoculation perceptible on the following day. In the very large majority of cases there was marked local and general reaction. The effects of this inoculation were manifest in from two to five hours. There was a feeling of malaise and faintness, with, as a rule, a rise of temperature of 2° to 4°. In some cases there was an actual rigor. At the point of inoculation there was marked pain and swelling. The feverish condition lasted, as a rule, for twenty-four to thirty-six hours, after which the local swelling diminished. In some cases there was marked enlargement of the lymphatic glands nearest to the point of inoculation which persisted in one case for weeks. In the cases where inoculation was performed twice, the second reaction was not so severe as the first.

Amongst the staff of the hospital, of the 58 inoculated, 9 developed enteric fever. Of those 9 cases one proved fatal, death supervening on the twenty-fifth day from a sudden attack of cardiac failure. It must in fairness be noted that the patient started his attack with an impaired heart from an old standing mitral lesion. Of the remaining 8 cases, 3 may be described as very severe attacks with persistent high fever, and marked abdominal or nervous symptoms. In 4 cases

the attack was pronounced, in one it was of an apyrexial type.

Relapse occurred in one case, there were complications in two cases; in both, pleurisy with effusion; in one, thrombosis of the veins of the leg.

Inoculation was carried out twice in the fatal case and in four of the milder attacks; once in the three very severe cases and in the apyrexial case.

Of the soldiers admitted into hospital who had been inoculated, inoculation had been performed once in every case. Eighteen cases of enteric were observed. Two were very severe, five severe, and ten were apparently modified attacks. Though the signs and symptoms of enteric fever were present they were very mild, the fever not being high and often of short duration (seven to ten days). In two complications occurred, five cases were admitted with a history of a slight fever with gastro-intestinal disturbance. It is impossible to be certain if these were cases of enteric fever. They may have been modified attacks, but some of the signs and symptoms were absent; and as they had been inoculated, the presence of a positive Widal's reaction was valueless from a diagnostic point of view.

Our experience thus has been that while inoculation *appears* to modify the disease, as completely modified attacks are met with in the uninoculated. Again, very severe attacks with complications and relapse occur in those who have been inoculated. One cannot from this conclude that inoculation has been valueless, for had not the patient been inoculated the attack might have been still more severe.

It is interesting in this relation to consider the significance as regards immunity indicated by the presence of an agglutinative reaction in the blood. On this point there is much difference of opinion. Some observers hold that agglutination and immunity are distinct; others that agglutination is the essence of immunity. Experimentally it is found that a high degree of agglutinative power is never present without a certain degree of immunity. It is probable that the presence of agglutination indicates that reactive processes have occurred in the body which lead to increased resistance.

Our experience of the presence of agglutination after anti-typhoid inoculation was very varied. In some individuals the

agglutinative power of the serum disappeared very rapidly, even in those who had been inoculated twice. In others who had been inoculated twice under the similar conditions there was marked agglutinative power after six months : the agglutinative power was indeed so high that the reaction took place almost instantaneously. Were these persons immune against enteric infection? I do not think one can go the length of saying that they possessed complete immunity against natural conditions of infection, but I think we can say that a reaction had taken place which led to an increased power of resistance.

It is exceedingly important to note that in the case of three of the staff who developed enteric fever, agglutinative power was *absent* during the early days of the fever, therefore we can with justice conclude that it was not present at the time of infection.

The effect of inoculation on the mortality of the disease can only be arrived at, I should consider, after years of careful observation, and from statistics carried over a long period and dealing with very large numbers. I think the outlook for inoculation is hopeful. Immunity is a relative term. While we cannot consider an inoculated person immune, I hope that we can consider that he has got an increased power of resistance.

An important question in relation to inoculation is, can it be considered entirely free from harmful results? With regret, I must state that it cannot. I have seen at least one case where inoculation markedly lowered the health standard of the individual. He was never well after his inoculation, and ultimately developed enteric fever some months afterwards.

The mortality in the cases under our care during the whole of the attack, was 10·7 per cent. Of these, two cases started their fever with a damaged heart, and died of cardiac failure. Perforation occurred in three cases. One was admitted perforated with a view to operation, but was found too near death for any operative measures to be undertaken. One case was operated on a very few hours after perforation, but at the time of operation he was very ill with dilated right heart and œdema of the lungs, and gradually sank and died. Of the remaining cases one died of hæmorrhage, one of bronchitis 19 days after tracheotomy for œdema of the glottis, and the rest from general

exhaustion from the severity of the attack and the pronounced toxæmia.

Dysentery in South Africa is a fairly common disease, of it we saw some forty cases. Of these a number were of the catarrhal variety. About half were of the more severe type. It is interesting to note the rarity of the amœba coli in the cases. We thought we found the parasite twice, but could not be absolutely certain. This experience is in line with that of other observers in South Africa. As regards treatment, one would say that if the case is of the mild or catarrhal type, sulphate of magnesia or castor oil and chlorodyne are the best remedies. If the case is of the severe type, the question is more difficult. One would not then advise sulphate of magnesia. In some cases it seemed that its use was only stopped before it had hastened the demise of the patient. In some cases, ipecacuanha *with, not without* ematin, seemed to act like a charm. In one or two cases no drug seemed to have any influence, and the condition only improved when all drugs were stopped by the mouth and morphia given hypodermically.

Of the general cases in hospital one might dilate for hours, but there is only one more point which might be brought before the Society to-night. It is the condition which we came to describe as the "soldier's heart." A large number of these cases came under observation. It was found in men who had been engaged with arduous marching for months on end. There was *no* history pointing to a previous valvular defect, and no anterio-sclerosis or kidney lesion. There was complaint of shortness of breath and general weakness. The area of cardiac dulness was increased laterally, especially on the right side. There was a systolic murmur present, with its point of differential maximum intensity at the tricuspid area, a venous pulse in the neck, and apparently no mitral lesion. I take it that there was dilatation of the right side of the heart from over strain, and consequent tricuspid regurgitation.

I hope that I have not unduly trespassed on the time of the Society, but I feel that some of the points noted are well worthy of thought and discussion, and I hope to gain information by bringing them up to-night.

Meeting V.—February 6th, 1901

Mr A. GORDON MILLER, *President, in the Chair*

The President made the following reference to the death of our Beloved and Revered Queen :—

Gentlemen,—Since our last meeting our nation has sustained a grievous and irreparable loss in the death of our beloved Queen. I am sure that I express the feelings of all the members of this Society when I say that we deeply deplore the death of the Queen.

When the history of the reign of Queen Victoria is written, prominence will be given to the great progress of Medical Science. It will be recorded also that during her reign the highest honour ever conferred on a member of our profession has been bestowed on our Honorary Member and former Vice-President, Lord Lister.

The Queen always took a deep and intelligent interest in things connected with our profession, especially in hospitals and in nursing, and we have with us the Victoria Ward and the Jubilee Pavilion in the Royal Infirmary, and the Queen Victoria Jubilee Institute for Nurses, to remind us of our good Queen's interest in all such philanthropic work. What, I think, best represents the nation's sentiments towards our late Queen is expressed in the following words of the apostle Paul, "I thank my God upon every remembrance of you."—*Phil.* i. 3.

He moved that an address of condolence and congratulation be drawn up and presented to His Majesty, King Edward, which was unanimously agreed to.

I. ELECTION OF MEMBERS

The following were elected Ordinary Members of the Society:—Noel D. Bardswell, M.D., M.R.C.P.Ed.; M.R.C.S.Eng.; and Stuart M'Donald, M.B., C.M., M.R.C.P.Ed.

II. ORIGINAL COMMUNICATION

GASTRIC HÆMORRHAGE AND ITS SURGICAL TREATMENT

By A. W. MAYO ROBSON, F.R.C.S., Senior Surgeon to the General Infirmary at Leeds, Emeritus Professor of Surgery in the Yorkshire College of the Victoria University

MR PRESIDENT,—I can assure you that I feel it a very high honour to be asked to address so distinguished a society as the Edinburgh Medico-Chirurgical; and as the northern capital has for ages been such a stronghold of medicine, I should almost have hesitated in venturing on another trespass of surgery, had I not felt that the opinion held by many practitioners of medicine "that bleeding from the stomach, the result of ulceration, rarely proves fatal," requires carefully reconsidering, and the facts remarshalling and looking at also from the surgical point of view.

Just as in other internal hæmorrhages, bleeding from the stomach was until quite recently a subject that, so far as treatment is concerned, only came under the notice of the physician; and it is only since the advances in surgery have demonstrated the feasibility and safety of exploring the abdomen, that the question of the surgical treatment of hæmatemesis has been a subject that could be discussed with advantage in a mixed medical and surgical society.

Were it desirable, the time at my disposal this evening would render it impossible to consider all the varieties of hæmatemesis. I shall therefore confine my remarks altogether to the most common, and certainly the most important cause of gastrorrhagia, simple ulcer, leaving out of consideration the vomiting of blood from cirrhosis of liver, cancer of stomach, miliary aneurism, aneurism of aorta and other vessels, purpura, scurvy, heart disease, leukæmia, typhoid fever, and other more obscure conditions only amenable to medical treatment.

When it is borne in mind that gastric ulcer occurs in 5 per cent. of the community, and that, according to Brinton, Habershon, Müller, Dreschfield, Lebert, and others, the mortality from all causes in all cases of ulcer is from 10 to 50 per cent., it must be at once allowed that the consideration of the subject is

a matter that should claim the attention of the profession to a greater extent than it has hitherto done.

Its importance will be better grasped if these figures are applied to the population of a city like Leeds, which, for convenience, I will take at 500,000. Of this number, 25,000 may be supposed to have or to have had ulcer of the stomach, and out of these, taking the mean estimate of mortality at 25 per cent., though this is under the mean as given by the authorities named, 6250, or 12,500 per million of the population, may be expected to die from gastric ulcer or some of its complications.

When it is fully grasped that many of the complications can only be dealt with successfully by surgical means, and that even in the mildest cases of gastric ulcer, some of the most serious complications, such as perforation and hæmorrhage, may suddenly supervene, I think that my audience will agree with me when I express an opinion that physicians and surgeons should in the treatment of these serious cases be in closer touch and have a more perfect understanding, so that valuable time may not be lost when surgical interference is required.

At present I have no hesitation in expressing my conviction that the treatment of gastric ulcer, especially among the working classes, is altogether too careless and unscientific, and I feel sure that the out-patient treatment of such cases, whether in hospital or private practice, is absolutely wrong, and attended with great danger, either of sudden catastrophe from rupture or bleeding, or of the transformation of an acute into a chronic disease, which can then only, in the greater number of cases, be set right by a surgical operation.

I think that as soon as gastric ulcer is suspected, the patient should be told the serious nature of the disease and the uncertainty of cure without rest in bed and long continued care in diet under proper medical supervision. If then, from business or other reasons, the advice of the medical attendant is ignored, the blame rests on the patient and not, as at present is so often the case, on the slipshod method of aiming at relief and calling it a cure.

Fortunately the surgical treatment of chronic and recurring gastric ulcer and its complications is becoming more and more advocated, and with increasing experience more and more perfect, so that to-day the tale is very different to what it was even a year ago, when I gave a series of lectures on the subject from

the Hunterian Chair at the Royal College of Surgeons of England. I was then able to collect from all sources 188 cases of operation for chronic gastric ulcer, including thirty-four of my own, but excluding operations for perforation and hæmorrhage; out of these 31 died, giving a mortality of 16·4 per cent.; but my recent experience in a largely extended number of cases shows barely a mortality rate of 5 per cent., and as all the patients were either chronic invalids or ill unto death, that means a saving of life in 95 per cent. of the cases, either by gastro-enterostomy, gastroplasty, pyloroplasty, pylorectomy or gastrolysis.

I must now confine myself to my special subject for this evening, the complication of hæmorrhage, which, according to different authorities, occurs in a greater or less degree in from 50 to 80 per cent. of all cases of gastric ulcer, and is, according to various authors, fatal when medically treated in from 2 to 3 per cent. (Müller 2 per cent., Welsh 3 to 5 per cent., Brinton 5 per cent., Debove 5 per cent., Steiner 6·36 per cent., Lebert 3 per cent., Dreschfield 3½ per cent., Rodman 8 per cent.). If we take the average, 7 per cent. of deaths in cases of gastrorrhagia from ulcer will not be far from the truth. If this is worked out in the 500,000 population of a city like Leeds, it means that of the 25,000 persons who have or have had ulcer, 12,500 suffer from hæmatemesis, and of these 7 per cent. or 875 die.

We are therefore dealing with a very serious and common accident, and one which a society composed of experienced physicians and surgeons such as are here assembled can do real good by discussing.

The bleeding may be arterial, venous or capillary, and curiously, capillary hæmorrhage may be so free as to render it difficult to say that some large vessel has not given way, yet after death a careful examination may fail to discover any gross vascular lesion.

Although capillary hæmorrhage may supervene on other conditions, such as congested portal circulation, enlargement of the spleen, and general engorgement of the venous system, when general treatment will usually be all sufficient, yet there are two special conditions in which the bleeding may be so free as to raise the question of surgical treatment: these are vicarious hæmatemesis at the menstrual period, and post-

operative hæmatemesis. The former condition is well recognised and usually yields to medical treatment. Petersen's experience of surgical treatment in three cases terminating fatally after operation would not encourage surgical intervention unless the circumstances of the case were very exceptional.

In my Hunterian Lectures, I drew attention to post-operative hæmatemesis, of which I have seen several cases, three fatal. The cases occurred after operation for intestinal obstruction, tubercular peritonitis and herniotomy, or after gall-bladder operations. Other cases have since been reported: one by Dr Winslow in a stout woman of 65, after operation for umbilical hernia, where death occurred from profuse gastrorrhagia on the third day, and at the *post-mortem* examination, though the stomach and intestines contained a large quantity of blood, no vascular lesion could be discovered; and another by Mr Mansell Moullin, in a young man of 23, who died from profuse hæmatemesis within forty-eight hours of a simple exploratory incision in the left iliac fossa. It was then neither generally recognised nor well understood, but since that time several papers have appeared on the subject both at home and abroad, and though now fully recognised its cause remains obscure. Herr Eiselberg tried to explain it as a result of injury to the omentum from several cases observed in the Konigsberg Klinik occurring after hernia operations, after operations involving ligature of the omentum, and after torsion of omentum. Although this explanation may serve as a cause, it does not account for all cases, as proved by one of my own, in which a malignant tumour of the common bile duct causing jaundice and distended gall-bladder was operated on under cocaine, the distended gall-bladder being simply exposed through a small incision, stitched to the parietes and drained, without in any way exposing any other viscera, yet violent hæmatemesis set in the day after operation and proved fatal in three days.

This case also disproves two other theories that have been advanced: one that general anæsthesia is the cause, and another that sepsis affords an explanation; the latter being a theory suggested by Dr Rodman in his oration before the American Medical Association in June 1900.

That sepsis was not the explanation in the only three cases

in which I have had the opportunity of observing the conditions after death is undoubted, for in each there was no sign of peritonitis or other septic trouble, and moreover there was no gross evidence of ulcer or injury to any vessel of sufficient size to be visible to the naked eye.

The theory of sepsis is also negatived by recovery in the greater number of cases, and by there being no sign of distension or of the other usual concomitants of septicæmia when it occurs after abdominal operations.

The only explanation that seems to me at all feasible is that the hæmorrhage is dependent on a reflex nervous influence, and it is quite possible that this same explanation applies to vicarious hæmatemesis at the menstrual period; but it should also be remembered that slight erosion of the mucous membrane of the stomach sufficiently deep to cause severe gastrorrhagia may be almost imperceptible to the naked eye, even when searched for carefully on the *post-mortem* table.

Though serious it is not necessarily fatal, and I have known several cases to yield to general treatment such as high injections of hot water at a temperature of from 112° to 116° as advocated by Tripier, free purgation by calomel, the stopping of all mouth feeding, and the application of ice to the epigastrium, etc.

In post-operative hæmatemesis, operation can be seldom feasible or advisable, and Reichard's experience of operations followed by death in two cases would lead me in the future as in the past to rely on general rather than on surgical treatment.

Venous hæmorrhage, if from perforation of one of the large trunks such as the coronary, splenic, or portal, may cause rapid death; but owing to the diminished pressure in the veins and the more languid circulation, a rapidly fatal result is less likely than in arterial bleeding, and should the hæmorrhage persist or recur, such cases would be likely to benefit by surgical treatment.

Arterial bleeding is mostly responsible for the serious and fatal hæmatemesis due to gastric ulcer. The bleeding may be from the small arterioles which radiate between the peptic glands, from the trunks along the curvatures, the coronary being the most frequently affected, or from extrinsic arteries, the splenic alone being responsible according to Brinton for 55 per cent. of the deaths in fatal hæmatemesis.

The following analysis of 54 fatal cases of hæmatemesis is taken from Savariaud's Thesis :—

Ulcerations of the splenic artery	17	cases.
„ „ coronary artery	6	„
„ „ pancreatico-duodenal	7	„
„ „ gastric arterioles	10	„
„ „ branches of the coronary vein	2	„
„ „ other veins	2	„
Vessel not determined	2	„
No vascular orifice visible	4	„
Vessel not mentioned	4	„

The pancreatico-duodenal and right gastro-epiploic are the arteries chiefly affected in pyloric and duodenal ulcers.

The peculiarly fatal nature of hæmorrhages from medium-sized arteries depends on the fact that an ulcer makes a lateral opening into the vessel which prevents natural hæmostasis by retraction and contraction of the artery. The whole force of the heart is then exerted on the clot which tends to form during the syncope that usually occurs when a large quantity of blood is suddenly poured out.

The interval between the first onset of bleeding and death is of great importance surgically, and is well shown in the table from M. Savariaud's Thesis, which is therefore worthy of note.

Vessel.	No. of Cases.	Sudden Death.	Rapid Death.	Survived Considerable Time.
Heart	4	1	1	2 (3 days)
Aorta	2	0	1	1 (10 days)
Hepatic	2	0	1	1 (10 days)
Splenic	17	3	7	7 (2 to 8 days)
Coronary	6	1	3	2
Pancreatico-duodenal	6	1	3	2 (8 to 15 days)
Arterioles	10	1	1	8 (4 to 15 days)
Small veins	4	1	1	2 (7 to 11 days)
Invisible veins	3	0	1	2 (21 days)

It shows that in the present state of our knowledge it is impossible to diagnose the size of the vessel perforated either from the amount of blood lost or the length of survival.

A probable diagnosis of the site of the bleeding may, however, often be arrived at in hæmatemesis from ulcer, by a careful study of the previous history, especially with regard to

the site of pain, the time of onset after food, and the influence of posture on it, as well as by the way in which the pain radiates. The situation of the tender spot, the presence or absence of tumour and of dilatation or contraction of the stomach also afford assistance.

As will be seen later, the diagnosis of the site of the ulcer, and therefore presumably of the bleeding, is of great importance from a surgical point of view, and will influence both the advice given as to treatment and the method of procedure when the abdomen is opened.

Treatment

From the fact that medical and general treatment is successful in arresting hæmatemesis in 93 per cent. of cases, and that it is difficult in the present state of our knowledge to say at first that the bleeding is not occurring from capillaries or small arterioles, it necessarily follows that medical treatment should always have a fair trial in every case of acute hæmatemesis. The very fact of medical treatment being so often successful in cases of apparently alarming hæmatemesis goes to show that capillary oozing or bleeding from arterioles is much more common and accounts for many more cases of gastric hæmorrhage than has hitherto been supposed; but while thoroughly believing this, we must also not close our eyes to the experience we have in general surgery of bleeding from medium-sized arteries, such as the radial or ulnar, which we know would rapidly bleed a patient to death, if only perforated on one side and surrounded by warm compresses, a condition that practically applies in all cases of hæmatemesis where the larger vessels are eroded.

If, therefore, medical treatment and rest properly carried out are not successful in arresting the bleeding in a few hours, or if after being arrested it recurs, we should be driven to the conclusion that a large vessel is perforated; and if a surgeon has not been previously asked to see the case, I would say emphatically that a surgical consultation ought to be held with a view to considering the question of operation and immediate arrest of the bleeding by direct treatment if the patient is in a fit condition to bear it, just as would be the case if an ovarian artery were bleeding in a case of ruptured extra-uterine gestation, or a brachial or radial after an accident.

The views I am enunciating to-day are perhaps more pronounced and more emphatic than those I expressed a year ago in my Hunterian lectures, this being the result of a much extended experience in the surgery of the stomach, a much greater success on all hands in the surgical treatment of stomach diseases, and a careful study of more recent statistics.

An analysis of the table by M. Savariaud of fifty-four cases showing the prolongation of life in various fatal cases of hæmatemesis, demonstrates that in ten out of fifty-four, that is in 18·5 per cent., death occurs so rapidly that there is barely time to consider the question of surgical treatment, and that in seventeen out of the fifty-four, which equals 31·4 per cent., death occurs within from twenty-four to thirty-six hours after the first appearance of hæmorrhage. Now although at present these cases are often allowed to die without operation, yet if the bleeding were from a radial or tibial, or any accessible artery, they would all be saved by the simple operation of direct ligature of the bleeding vessel, which operation would also cure the bleeding stomach artery could it be readily found. The difference between the two is, however, not only one of accessibility, but also one of diagnosis; for if we are able to diagnose capillary hæmorrhage, the case will be generally one for medical treatment alone, but if the time arrives when we can diagnose with great probability arterial hæmorrhage from a large vessel the case will, I believe, be considered one for the adoption of surgical measures.

Our efforts both as physicians and surgeons must therefore be directed towards making a diagnosis of the size of the damaged vessel, for if it can be asserted that in all probability a large artery has given way, an operation could be undertaken in an early stage of the case before exhaustion and profound anæmia have supervened, with great probability of success.

Where there have been distinct signs of gastric ulcer preceding the hæmorrhage, and where a sudden hæmatemesis has occurred with great loss of blood accompanied by an attack of syncope, a large vessel will usually be found to be the source of the bleeding. In all such cases not speedily yielding to medical and general means, surgical treatment will probably in the future be carefully considered, and in some it will be followed out, for there can be no absolute rule formulated that will apply to every case, and each must be considered on its

merits; the present condition of the patient, the previous history, the surroundings, the possibility of skilled surgery and of good nursing and other circumstances will all help in the decision.

Although both surgical and medical treatment in cases of fulminating hæmorrhage have so far yielded disappointing results, in the remaining 50 per cent. of fatal hæmorrhages, where repeated bleedings occur and the interval between the first seizure and death varies from a few days to two or three weeks, medical treatment will have been fully tried and failed, and there can be no question as to the advisability of surgical procedures being adopted.

At present, with the exception of Dieulafoy, who advocates operation during the first bleeding if as much as half a litre of blood is lost, all other surgeons who have written on the subject agree that general means ought to be relied on during and after a first attack, as in from 93 to 95 per cent. of cases such treatment succeeds, and until our means of diagnosis as to size of vessel injured is rendered more reliable we must assent to this rule; but after a second bleeding, I have no hesitation in advising operation even during the course of the hæmorrhage if the patient can stand it, or as soon after as his condition will permit the operation to be done, for experience tells us that further hæmorrhages are almost certain to occur unless preventive measures be adopted. I saw a case of this kind last year which created a great impression on my mind. I was asked to see a single woman, close on thirty, with a view to operation after a second attack of hæmatemesis, which had, however, ceased when I saw her; and as the patient, though anæmic, was apparently much easier and expressed herself as feeling well, operation was deferred, and a third attack, which occurred a few days later during my temporary absence from home, proved fatal.

In another case, I operated during the third attack, found the bleeding vessel, and saved the patient.

Doubtless the desirability of stopping bleeding from an ulcer of the stomach by surgical means must have occurred to many minds before being put into execution.

To Mikulicz, however, belongs the claim of having been the first to operate for hæmorrhage from a gastric ulcer on February 13th, 1887, though Roux, of Laussaune, was the first to perform a successful operation, and that in a case of hæmorrhage

from the coronary artery, in which he excised the ulcer and ligatured the artery at both ends. In my Hunterian lectures I drew up a table of all the cases of operations for hæmatemesis I could then find. It included operations for post-operative and vicarious hæmorrhage, cases not associated with ulceration and all ending fatally. Excluding these, and correcting Hartmann's series, it would give 26 cases of acute hæmatemesis with 14 deaths, and 19 cases of chronic hæmatemesis with 2 deaths, otherwise a mortality of 53·8 per cent. in acute and of 10·5 per cent. in chronic hæmatemesis treated surgically. A more recent table has been drawn up by Dr Rodman (*Philad. Med. Jour.*, June 9, 1900), in which he has collected 32 operations for acute gastrorrhagia with 13 deaths, or a 40·6 per cent. mortality, and 31 operations for frequently recurring or so-called chronic hæmorrhages with 6 deaths, or a mortality of 19·3 per cent.; and even since that list was drawn up, other operations have been performed. This mortality is very high, but it must be remembered that in every case medical treatment had been first tried, and in many of the cases probably over too long a period before operation was performed: for instance, in one of my cases in which I operated successfully during the third seizure, the blood seen at the operation was so diluted and thin as to be more like water tinged with blood than blood itself, yet the bleeding was still going on. Moreover it is to be remembered that gastric surgery is advancing in safety and efficiency by leaps and bounds, and will before long, I feel sure, be one of the most satisfactory branches of the surgeon's work.

Technique of Operation for Gastrorrhagia

On account of the fear of exciting fresh hæmorrhage, it is undesirable to wash out the stomach before operating on a bleeding ulcer, and as the patient is usually anæmic and will therefore bear shock badly, it is desirable to have him enveloped in cotton wool or otherwise kept warm on a heated table, and to give him a subcutaneous injection of strychnine before operation. If it be unnecessary to infuse saline fluid before the operation, an assistant should be ready to perform that duty while the abdominal operation is being proceeded with should it be called for. After the abdomen is opened, the stomach may be emptied by pressing the contents into the duodenum,

as suggested by M. Terrier. This saves much time, and rapidity is of great importance. The surface of the stomach should then be carefully examined, as very frequently a puckering of the surface or thickening of the coats will be noticed at the site of the ulcer, or a difference in colour may sometimes indicate its site. Should the operator be fortunately able to find these indications much time may be saved, but if no indications of this sort be obtainable, the stomach must be drawn well forward and opened either by a vertical incision, which gives less hæmorrhage, or by an incision in the long axis of the stomach, which affords a much better view of the interior. In order to avoid soiling the peritoneum, thin flat sponges should be placed within the abdomen, and it is well to have a perforated sterilized gauze pad covering the stomach, the opening to correspond with the opening in the stomach. The edges of the stomach should then be held open by forceps, one above and below and one at each end.

The interior of the stomach must now be systematically examined, first the anterior and posterior surfaces, then the upper and lower curvatures, and lastly the pyloric and cardiac orifices, all of which can be done by the naked eye or by the aid of an electric cystoscope or a reflected light. If the posterior wall of the stomach cannot be efficiently examined, a slit may be made in the omentum and two fingers, or even the whole hand, pushed through it and employed to invaginate the posterior wall. If no ulcer can be found on the wall of the stomach the duodenum should be explored, not only by the finger, but by invagination. If no ulcer be found anywhere and the bleeding proves to be capillary or from small undiscoverable ulcers, gastro-enterostomy should be performed; but if an ulcer be discovered and it be possible to excise it, that operation should be done, as it undoubtedly offers the likeliest method of cure. After excision of the ulcer and ligature of the bleeding vessels the edges of the wound are brought together by sutures, of which one or two layers may be employed. Should the excision involve the peritoneal coat, it is desirable to apply Lembert's sutures; and if it be on the posterior wall, the operation may present some, though not insuperable difficulty. Should the ulcer be discovered in some region where excision is impracticable—for instance, near the cardiac end—the method adopted by Mikulicz, in his first

operation in 1887, of applying the cautery, may be employed. This seems to have answered well in several cases, and where death occurred afterwards it was not from a recurrence of the bleeding. In a case of this kind, gastro-enterostomy is also advisable by way of setting the stomach at rest. If any superficial bleeding points be discovered they may be caught up by forceps and ligatured, but this is more easily said than done, as the mucous membrane is very friable, and a ligature if drawn at all tightly tends to cut its way through; moreover, forceps easily tear the mucous membrane and may produce a larger bleeding area. The method of ligature *en masse* may be employed if the ulcer is small; it has proved serviceable in several cases, for instance in Case V., which will be described presently, and it is easy of application when the ulcer can be reached. It has been tested by experiments on animals by Dr Eisendrath, but has been shown to endanger perforation if the serous coat be taken up, except when supported by external sutures. Ligature of the main arteries of the stomach was suggested theoretically by Savariaud when no bleeding ulcer could be discovered; it does not, however, commend itself as being safe or likely to be efficient.

Should the bleeding be from an ulcer at the pylorus, the operation of pyloroplasty may be performed, the ulcer being excised and the edges of the gap left by the excised ulcer as well as the edges of the incision being united, so that the line of sutures shall be transverse to the axis of the pylorus. I would not, however, advise pyloroplasty when there is active ulceration of the pylorus unless the ulcer can be completely excised, for in three cases of this kind I have had subsequent disappointments owing to contraction in healing. In one case the patient made a good recovery from the operation but subsequently suffered from stenosis, for which a further operation had to be done. In a second case, recovery took place, and the patient regained his lost weight, but subsequently a tumour of the pylorus developed which was thought to be cancer and for which I had to perform pylorectomy, and in the third case perforation subsequently occurred. I think, therefore, that in cases of ulcer of the pylorus, where a clean excision of the ulcer cannot be done, it would be better to perform gastro-enterostomy or pylorectomy, whichever at the time may appear to be easier.

Wherever the pylorus is adherent, as in my experience it is in so many cases of ulcer, I should certainly prefer gastro-enterostomy; but if the pylorus be extensively ulcerated and free from adhesions, pylorectomy can be almost as quickly performed as gastro-enterostomy, seeing that the pylorus is already in the hand. Although the operative treatment of parenchymatous or capillary hæmorrhage by any surgical method would seem to be of doubtful value, for according to a report by Petersen from Czerny's clinic of three cases operated upon for severe parenchymatous hæmorrhage from vicarious menstruation, operation entirely failed to relieve, and of two other cases of post-operative parenchymatous hæmorrhage by Reichard, both died, yet if such cases, owing to persistence seem to call for operation, then gastro-enterostomy, by securing complete physiological rest, would seem to offer the best chance of success.

In performing gastro-enterostomy my recent experience has led me to prefer the posterior operation, which I have modified by using my decalcified bone bobbin as a hollow splint over which to apply the double continuous suture uniting the mucous and serous margins of the openings between the viscera, thus securing an immediately patent channel between the stomach and jejunum and a temporary protection to the line of union.

I may say that my last twenty gastro-enterostomies by this plan have all recovered without regurgitant vomiting or other untoward symptoms, thus affording a great contrast to what has occurred in the anterior operations both in my own and the hands of other surgeons.

The conclusions I would urge concerning the treatment of gastric ulcer are that—

(1) All cases of acute uncomplicated gastric ulcer should be submitted to thorough medical treatment in the shape of long-continued rest and attention to diet, the cases not being allowed to get up or to resume solid food until at least a fortnight after all disappearance of pain.

(2) Where the ulceration persists and proves intractable to medical treatment, or where relapses occur, gastro-enterostomy should be performed, so as to secure physiological rest and relieve the hyperacidity of the gastric juice nearly always found in such cases.

(3) Perforation demands immediate surgical treatment.

(4) The complications of disabling adhesions around the stomach and pylorus, pyloric contraction and hour-glass contraction due to chronic ulcers leading to pain, dilatation, loss of flesh and general impairment of health, and now often treated as chronic indigestion, should be treated surgically.

(5) In recurring, or so-called chronic hæmatemesis from gastric ulcer, surgical treatment is decidedly called for.

(6) In acute hæmatemesis, further accuracy in diagnosis as to the size of the bleeding vessels is urgently needed; and the co-operation of the physician and surgeon is advisable in all cases of hæmatemesis, so that if relief be not obtained by medical and general treatment, surgical means may be adopted if the bleeding is believed to occur from a large vessel; but seeing that capillary hæmorrhage is capable of relief by medical means alone, medical should always precede surgical treatment.

The following are brief descriptions of all the cases of hæmatemesis on which I have operated:—

CASE I. Chronic hæmatemesis—ulcer of pylorus—pylorectomy.

In 1891, I was asked by a medical friend to see a man of 54, who for six months had suffered from pain coming on an hour after food, and more recently from vomiting of blood of coffee-ground character in considerable quantities, so that he was not only reduced in flesh and strength, but had been rendered profoundly anæmic by the loss of blood.

A tumour of the pylorus could be easily felt, and the stomach was markedly dilated.

As he was rapidly losing ground I operated, and found the pylorus thickened and nodular, with adhesions to the liver and omentum.

After separating the adhesions, the pylorus was excised and the open end of the duodenum was fixed to the opening in the stomach, the rest of the stomach aperture being closed by a double layer of sutures.

The tumour proved to be inflammatory around an open ulcer which had been the source of the hæmorrhage. The bleeding was not repeated, and the patient rapidly gained flesh and returned home within the month.

CASE II. Hæmatemesis following on stab wound of abdomen—abdominal section.

On January 27th, 1897, a young mechanic, aged 20, was admitted to the Leeds General Infirmary, under my care, with free vomiting of bright blood following on a stab wound of the abdomen, the wound having been inflicted by a long, fine, triangular file, that had caught in the lathe at which the youth had been turning.

On admission he was pale and collapsed, with a pulse of 190, and as there were signs of free fluid in the peritoneal cavity, it was thought that the stomach had been perforated, and that the bleeding was from a ruptured gastric artery.

On opening the abdomen a large quantity of blood and clot was found free in the abdominal cavity and in the kidney pouches and pelvis, but on examining carefully the stomach no perforation could be found. After some searching the superior mesenteric vein was found bleeding freely, the file having bruised and pushed aside the stomach, pierced the great omentum and then wounded the vein.

The abdomen was washed out with hot water after the vein had been ligatured, and this also arrested the gastrorrhagia, as the patient made a good recovery and had no further hæmatemesis. The shock was relieved by the infusion of three pints of normal saline solution into the median basilic vein and by injections of liq. strychniæ.

CASE III. Acute hæmatemesis from ulcer of duodenum eroding gastro-duodenal artery—anterior gastro-enterostomy with Murphy's button.

I was asked by my colleague, Dr Churton, to see a man, aged 36, who after a year's history of ulcer of the stomach with great loss of flesh and strength, was suddenly seized with profuse hæmatemesis, which was followed in twenty-four hours by a second seizure when I saw him. He was then extremely weak, and very thin and anæmic, and looked as if he must die if further hæmorrhage occurred. His pulse at the time of operation was 150.

The history clearly pointed to the pylorus as being the seat of bleeding, but the patient was too ill to bear pylorectomy, and an anterior gastro-enterostomy was performed by means of a Murphy button, the whole operation occupying only fifteen minutes. He bore the shock well, and for five days made good progress; on the sixth day he was suddenly seized with severe abdominal pain, and from that time he lost strength: vomiting

set in on the eighth day, and he died on the tenth. No further bleeding occurred, and at the necropsy a duodenal ulcer opening into the gastro-duodenal artery was found. The cause of death was peritonitis, the result of a leakage when the button began to separate.

So far as the hæmorrhage was concerned, the gastro-enterostomy had been completely successful, and the vessel, though perforated at one side, was occupied by a firm clot.

CASE IV. Mr C., aged 44, was sent to me by Dr Peter Macgregor of Huddersfield, on the 2nd of January 1900, with the history that he had had pain after food together with other symptoms of gastric ulcer for four years, though his first attack of vomiting blood occurred in May of the previous year. The bleeding was rather profuse, but under medical treatment and rest he recovered, and was able to resume his occupation. A week before he was sent to me there had been a recurrence of the hæmorrhage, which was repeated three days later.

On his arrival at the surgical home he was in a state of collapse with a barely perceptible pulse. There was well marked dilatation of the stomach with tenderness over the pylorus. The history of the pain occurring one and a half hours after food, together with the site of the tenderness, and the presence of a large amount of blood in the motions, left no doubt but that the ulcer was at or near the pylorus. As the bleeding had recurred twice in the week and was probably persisting and passing into the bowel, operation was decided on. This was done on January 4th. As the patient was in such a feeble condition every precaution was taken to guard against shock, and it was decided only to perform gastro-enterostomy, as it was quite clear that he would not stand any prolonged search for the bleeding point.

A posterior gastro-enterostomy was therefore done, a decalcified bone bobbin being used to make the anastomosis. His temperature and pulse were never above normal, and he made an uninterrupted recovery. He was fed by nutrient enemata for the first few days, only taking sips of water by the mouth, but at the end of that time he was allowed to take milk and soda water, and then gradually to have stronger food. He was up at the end of the third week, and returned home within the month. A year later he was in perfect health, and had had no recurrence of his stomach symptoms nor any further bleeding.

CASE V. On July 6th I was asked by Dr Humphery, of Armley, to see a patient, Mrs H., aged 33, who had been seized with violent hæmatemesis a week previously, the attack having recurred four days later, and again on the day that I saw her. The patient had been anæmic before operation, and had had some slight indigestion, but otherwise did not consider herself ill, and she had been married only three months. On my arrival I found her quite blanched, and with a very rapid feeble pulse, but as the bleeding had resisted ordinary remedies, and as she was clearly going to die if unrelieved, though it seemed a rather desperate procedure, we decided to give her the chance of relief by operation. She was removed by ambulance to a surgical home, and as during the journey the bleeding began again, I operated without delay. A pint of saline fluid containing one ounce of brandy was given into the rectum, and 10 minims of liq. strychniæ were administered just before the operation, the patient being completely enveloped in cotton wool. On opening the abdomen there were no indications on the surface of the stomach as to the situation of the bleeding ulcer. I therefore made an incision in the axis of the stomach, which enabled me to explore the interior. I counted no less than seven bleeding points. As two of them were bleeding freely, I took them up by artery forceps and ligatured the mucous membrane *en masse*, the other points stopped on exposure to air. I then rapidly closed the wound in the stomach, and performed posterior gastro-enterostomy, using one of my bone bobbins as a splint over which to make the anastomosis. We had a little anxiety on account of shock for the first twenty-four hours, but by administering saline fluid by the bowel and injecting it freely into the subcutaneous tissue, together with the administration of several injections of liq. strychniæ, the difficulties were tided over, and the patient's further progress towards recovery was uninterrupted. She was able to take solid food in the second week, and when she left at the end of the month, she could eat any ordinary food without discomfort. Six months later she had had no recurrence of symptoms, and was well in every respect.

Five is not a large number of cases to report, but the treatment of hæmatemesis by operation is a comparatively new operation, and only few cases have been reported. Had it not been for the accident of leakage when a Murphy button was

beginning to separate there would have been no casualty in the list, and that is an avoidable accident by employing sutures around a decalcified bone bobbin, which is now my regular custom. Undoubtedly without operation all the patients would have died. The cases present individual points of interest.

In the first case a pylorectomy was performed as the pylorus was the site of a tumour that might have been malignant, though could it have been asserted that the enlargement was simply thickening around an ulcer, gastro-enterostomy would have answered equally well as it did in the third and fourth cases, by securing physiological rest and freedom from the irritation due to hyperacidity of the gastric juice which is usually present in ulcer of the stomach.

In the second case, hot lavage acted in the same way that it does when given by high injections into the bowel, only in this case it was necessary to open the abdomen to search for and ligature the large vessel bleeding into the peritoneum.

The fifth case illustrated the feasibility of exploring the stomach even in a patient brought very low by excessive loss of blood ; and although the ligature of the chief bleeding points was effected, it was deemed advisable to secure rest to the stomach by performing a posterior gastro-enterostomy, especially as it could be done without materially prolonging the operation.

It only remains for me to thank you for giving me such a patient hearing, and to hope that the discussion which is to follow may prove of interest to the Society and useful to the profession at large.

DISCUSSION

DR AFFLECK, in opening the discussion, was sure that they had listened to the paper just read with interest, pleasure, and profit. The subject was one of great importance, and as they had listened to Mr Mayo Robson's narration of facts and cases, he was sure that they must have felt that the results which he had attained were to be ascribed not merely to surgical aptitude and dexterity, but quite as much to careful study and thought, to sound judgment, and to large clinical experience. It was difficult in the short time at his disposal to follow Mr Mayo Robson through the many important statements which he had made. His treatment of the subject was largely from the surgical point of view, so that he (Dr Affleck) could only speak

on one or two aspects of the subject. But perhaps a comparison of experiences, even though it were a comparison of small things with great, might not be altogether unprofitable; and particularly in a subject like this, where further information is desirable. The subject was one of exceeding interest both to physician and surgeon. Not long since it was only interesting to the physician, but it was a conspicuous feature of modern surgery that it had boldly entered into territories previously deemed inaccessible, and had grappled successfully with problems and conditions that were formerly regarded as hopeless; and speaking as one who had had some considerable experience in the surgery that comes to the aid of medicine—particularly the surgery of the abdominal cavity—he confessed to a feeling of constant gratitude to the science and art of surgery for what it had been able to do in co-operation with the physician in the saving of life and the relief of suffering; and he thought there was promise in the future of still more to be done by surgery for medicine.

With regard to hæmatemesis, and confining himself to gastric ulcer as a cause, although it arose from many other causes, he would make a few remarks, firstly, in reference to diagnosis, and, secondly, in reference to treatment. In regard to diagnosis, it was the case that no one symptom was an absolutely certain diagnostic sign of gastric ulcer, but there was often clinical evidence that was very valuable, and the physician might oftentimes be almost certain. There were one or two points which he might mention in connection with what Mr Mayo Robson had said as to being able to diagnose the character of an ulcer by certain symptoms. With regard to the pain, for instance, he was sure they could not always rely upon the evidence furnished by this symptom as to situation, acuteness, or chronicity of the ulcer. He found in his experience that the pain of gastric ulcer was rather misleading. It had been stated that when the pain arises immediately after the ingestion of food the ulcer is likely to be seated at the cardiac end of the stomach, but that if it comes on later, the probability is that it is at the pyloric end. He was not sure that they could attach very much importance to this, because he doubted very much if the pain was due to the contact of food with the ulcer, and not rather to the setting agoing of the active work of the stomach, the ingestion of food causing increased movement and the secretion of gastric juice in abundance. In this he might be wrong, but he had seen cases in which the *post-mortem* examination did not confirm what the symptoms suggested. He had a case, for instance, of severe gastric hæmorrhage where the patient complained of pain not in the epigastrium at all, but in one of the iliac fossæ. They knew the pain was often a referred pain, just as in the case of pleurisy

and others. Nor was the decubitus of the patient altogether to be relied upon. Still these were important points, and he thought Mr Mayo Robson had been fortunate in his diagnosis. The use of chemical investigation of the vomit and contents of the stomach was often, he thought, of very great value, and no physician could afford to ignore that line of investigation in doubtful cases. In the case of ulcers of long standing they had accessory evidence—alteration in the size of the stomach, sometimes large and dilated, sometimes an hour-glass contraction, the presence of pyloric tumour, etc., these were all helpful in diagnosis. The method of introducing tartaric acid and bicarbonate of soda for the purpose of coming to a diagnosis as to the size of the stomach was, he thought, questionable. If an ulcer were present it might lead to rupture, although he had not seen any accidents of this sort on record. He had frequently been able to diagnose size by a much gentler procedure. Gentle friction of the skin with the hand sometimes makes the stomach stand up quite sufficiently to enable it to be clearly mapped out. He had even seen hour-glass contractions of the stomach at certain points clearly mapped out in this way. Of course some chronic ulcers did not bleed, nor did all acute ones; but the chronic ulcer is generally known by the recurring hæmorrhage after a considerable time, and these were the cases which he thought were more suited for surgical treatment. The acute ulcers were characterised as a rule by the sudden onset of very violent hæmorrhage, occasionally fatal, not often immediately so, but by recurrences at intervals of a few hours over a day or two. A large number of these evidently undergo spontaneous arrest of the bleeding. With regard to treatment, he thought that in the case of acute ulcers they must trust largely to medical treatment in the first instance, though he wondered sometimes how far medical treatment contributed to the arrest of the hæmorrhage, or whether it really did not arrest itself. He had tried all manner of styptics, and had sometimes failed with them all. He thought morphia one of the best, along with absolute rest; and, in one of the worst cases he had met with, he had administered as a last resort very hot water, and that was followed by an arrest of the bleeding, whether due to this treatment or not he could not say. That most of these acute hæmorrhages undergo spontaneous arrest is a fact that the physician takes advantage of, and justifiably so, in trying to assure the patient and the patient's friends. He had found it often of the greatest service to be able to say that the bleeding was likely to stop in a short time, though of course we must not forget that a considerable number did prove fatal.

He was struck with Mr Mayo Robson's remarks as to acute gastric ulcer. He advocated that, after perhaps the second

hæmorrhage, surgical interference should at least be proposed if the patient's condition admitted of it. In hospital work particularly, he thought the patient's condition would often not admit of it. Yet it was possible that by timely interference we might save lives sometimes. He thought, however, they wanted more evidence. He had a striking case in his mind. A hæmorrhage had occurred in a young woman some years ago. She was brought into the infirmary in a state of collapse, the history being rather that of a chronic ulcer, and she brought up blood largely, and passed it by the bowel largely. All means that could be taken to arrest the hæmorrhage were unavailing, and I am sure that Mr Mayo Robson himself would have been satisfied that it was not a case for surgical interference. She lingered two or three days, and died. At the *post-mortem* he had found no fewer than twenty ulcers in the stomach, and four in the first part of the duodenum. They were not merely erosions, but ulcers that had broken through the mucous membrane. There was a clot in one of these ulcers; but on consideration, he thought the bleeding must have come from most of them, as there was copious hæmorrhage downwards as well as upwards. It was an exceptional case, but there were on record cases with even a larger number of ulcers. Of course, many acute ulcers that bleed largely underwent perfect healing. But even in the chronic ulcers, an effort at cure is made. It had been pointed out long ago that often a chronic ulcer might be seen where the cicatricial process was almost successful, but not quite. Some vessel is left open from which hæmorrhage takes place from time to time. These were the cases that seemed to call for surgical treatment. It was interesting to find that even in these cases a healing process was under way; but nature's efforts at healing a chronic ulcer were often very inadequate. The sequelæ of chronic ulcer were often the cause of a great deal of suffering, and in reference to these cases he was quite at one with Mr Mayo Robson. Patients might under treatment by rest, diet, etc., get apparently quite well; but were they well? They were not, and often came back again after years of suffering; it might be that life was curtailed. These cases also seemed to him to be specially suited for the intervention of the surgeon. In these chronic cases, however, much might be done by syphoning the stomach. He thought the introduction of this method of treatment of gastric disease was a great step in advance, and some patients would be content with the discomfort rather than face the formidable idea of surgical operation. They must remember that they had the patient to consider, who would often be more chary of submitting to operation for a malady of this sort than for a tumour or something which he could actually see. Surgical methods should

not be lightly undertaken, but he thought that in cases of this sort physician and surgeon should be in touch. Unless the surgeon had specially studied the clinical history and symptomatology of gastric ulcer, he should not be without the co-operation of the physician in the matter. In conclusion, he would say that in some of these severe cases of chronic gastric ulcer we should at all events entertain the idea of the use of surgery more than we were wont to do. He would mention a case in which he had been interested. In September 1899 he had a young woman with gastric ulcer under his charge in the Royal Infirmary. It was a chronic ulcer, and she had had hæmorrhage some time before admission. After treatment by rest and rectal alimentation, etc., she apparently got well, but he never felt satisfied that she was quite well. She resumed her occupation, and shortly afterwards went to Paris. While there, some six months afterwards, she was seized with a violent hæmorrhage. She was taken to a hospital in Paris, and seems at once to have been put under surgical care. The operation of gastro-enterostomy was performed. She came home as soon as able to leave the hospital, and came again to the Infirmary here. One would never have known her, she was so reduced, but under careful treatment she gained in weight, and left perfectly well. A wound was to be seen in the abdominal wall, but he could not have said what had been done had the surgeon not given the girl a little scrap of paper with a sketch of what had been done. He had perhaps taken up too much of the time of the Society, but he had a great interest in the subject which had been so ably placed before them by Mr Mayo Robson.

SIR WILLIAM GAIRDNER said that the first feeling he had after hearing Mr Mayo Robson's paper was one of intense relief that he was willing to leave still a little corner for the physician. He understood that the physician might be allowed to watch the case for a little until the surgeon should come in. From what was heard from some of their surgical friends he was not prepared for this much—the surgeons had been so aggressive of late years that it might be doubted whether there was any function for the physician at all. Take the brain for instance. What was a brain? they said. The physician had merely to say where the lesion was, and then to call in surgery. The kidney —well, the physician might fight with this for a little, but it ultimately came to this, that the surgeon had to be called in to remove the stone or remove the kidney. The stomach. What's a stomach? A thing that caused a lot of trouble; and when the trouble became too much—well, we could get on without a stomach, according to the modern surgeon. Mr Mayo Robson, however, did not go so far as that. He admitted that we have

some use for the stomach, and also that physicians have still some work cut out for them.

But he (the speaker) must confess that Mr Mayo Robson in his very remarkable appeal had shown that there was a great deal more to be done by surgery for the stomach than they had been accustomed to think; and on looking back, he would confess to remembering a good many cases where surgery might have saved a patient, and where the patient had perished for want of what surgery could do now. He remembered one case in which a woman was bleeding to death—he was fully alive to it, and wandered through a whole evening to find a surgeon who was willing to cut down and staunch the bleeding. He failed to get him, and the woman died. They were fortunate as a society in having been able to secure Mr Mayo Robson, and thankful to him for the admirable paper which he had read.

PROFESSOR GREENFIELD did not like to take up the time of the Society when one had not concentrated one's thoughts. He should like to have traversed Mr Mayo Robson's statistics if he had had the courage; but was aware of the extreme care and caution which Mr Mayo Robson exercised in these matters, and would not do anything of the kind without carefully looking into the subject. Like all his statistics, one felt that these had been compiled with the greatest care; but they did not seem to represent the results of one's own experience, and therefore required very considerable proof. There was not much hope, however, of reducing the large mortality indicated by medical figures, and therefore surgeons would have the larger scope and practice. He thought that physicians ought first to lay their heads together to come to some definite conclusion as to what were diagnostic points of ulcer in the stomach. In the difficult cases they should endeavour to help the surgeons by endeavouring to arrive with precision at the exact locality of the ulcer. The known incidence of ulcer in the stomach should be considered. He would go first to the position where we knew ulcers were most common. As to the cases where one would be thankful for operation—he thought duodenal ulcers came under this head—the risks of recurrence were very great. When situated at the pylorus or within two inches of the pylorus, not only is there risk of stricture, but healing is frequently faulty and the ulcer is liable to recur. If we can diagnose these, we ought to encourage rather than discourage, at anyrate, exploration; and, if possible, operation. The most difficult cases were perhaps those of the chronic ulcers which gave rise to none of the common symptoms. Some of these were where the ulcer was duodenal, or on the posterior wall of the stomach, burrowing deeply above the pancreas and perhaps eroding the splenic artery or vein. In reference to these cases

they might ask Mr Mayo Robson whether he has operated upon any cases in which the ulcer had formed a deep funnel-shaped erosion, possibly into the pancreas, as is sometimes the case. It would be an important point to know whether a plastic operation on the wall of the stomach is possible in such cases, and whether it should be attempted at the time of operation. He might say that he thought Mr Mayo Robson had hardly represented what was the usual practice in Edinburgh in the treatment of chronic ulcer. He did not think it was usual to allow these patients to go about—he should be sorry to do so—and he thought that a much more prolonged period of rest than a fortnight should be enforced (Mr Mayo Robson intimated that he had said a fortnight after the cessation of all discomfort and pain). The speaker understood Mr Mayo Robson to infer that these patients were allowed to go about and be treated as out-patients. He might add that he had been very much impressed in some cases of recurrent hæmorrhage with the value of the continuous use of large quantities of iron. He could recall several cases where the persistent use of large quantities of iron had been followed apparently by perfect cure.

DR JOSEPH BELL divided all medical authorities into two classes—one class whose books one never read, and the other whose books one read all that could be got hold of. Mr Mayo Robson was of the latter class. He entirely agreed with the views he had expressed in this paper. He was sorry to say that during the time he was surgeon to the Royal Infirmary no physician had ever asked him to open an abdomen for hæmatemesis ; and from his own lack of experience in the matter he would not occupy the time of the Society, but merely thank Mr Mayo Robson for his paper, which he had listened to with the greatest possible advantage.

MR CAIRD felt that they owed Mr Mayo Robson a debt of gratitude for the manner in which he had emphasised the early co-operation of physician and surgeon. It was formerly the case that the surgeon was summoned only when the resources of the Pharmacopœia had proved futile, and when, unfortunately, surgical measures were equally futile. The present increasing success and safety associated with surgical intervention gives one the greatest encouragement to interfere in serious conditions before it is yet too late. He would like to have followed Mr Robson into the various questions concerning the operation of gastro-enterostomy, etc., which had been touched upon, but he felt himself bound to adhere to the subject under discussion, that of the treatment of hæmatemesis, due to gastric ulceration.

Mr Robson's statistics as to the frequency of fatal hæmorrhage were very striking, and it would be interesting to learn the indi-

vidual experience of those present as to the relative frequency of fatal cases occurring in private practice on the one hand, and under the enforced rest and quiet of hospital care on the other. Personally he only knew of a single instance in the Royal Infirmary which terminated fatally in the hands of the physician. He gathered that the physician so frequently saw recovery from impending death in such cases, that he was generally sanguine as to the result of general treatment, and was therefore loath to avail himself of heroic measures of doubtful efficacy.

Mr Robson had divided the severe cases which required surgical aid, firstly, into those where a typical ulcer existed with arterial erosion, giving rise either to a fulminating or to recurrent dangerous hæmorrhage; and, secondly, into the type where lesions were practically microscopic, or of the nature of post-operative hæmatemesis.

Now ordinary surgical principles suffice for the arrest of hæmorrhage, no matter where, and hence in the fulminating type there is a clear indication to expose and secure the bleeding point. This is in itself, however, a serious procedure, and under the circumstances when it is demanded, may serve to hasten the end in spite of an after transfusion. Hence it might be asked if no simpler and safer, albeit less certain method, could not be accepted. Mr Caird thought that this could be done by enforcing the conditions of absolute rest so essential in the natural arrest of hæmorrhage. He thought if we looked upon the treatment of vesical hæmorrhage as analogous, and reflected upon the excellent results gained by incision and drainage, how frequently this prolonged life for many months, or again, how it abolished the immediate risk of death from bleeding, and allowed us thereafter to treat, and perhaps remove, the cause, then we might apply the same principles to the gastric organ. Moreover, the stomach is more advantageously situated for such procedures than the bladder, in so far as we can give a greater degree of rest.

He would therefore recommend in such cases of active gastric hæmorrhage the performance of a gastrostomy to give rest and drainage, and I would select the operation of Witzel or some of its modifications as being speedy, safe and readily performed with local anæsthesia if need be. This would permit one to empty the stomach, to drain off the continuous gastric secretion, and to recognise any fresh bleeding. The stomach thus kept empty vomiting would cease, rectal alimentation would be maintained, and should fresh hæmorrhage ensue, it could be checked by the use of hot saline lavage.

He would specially recommend this plan in the second series of cases for which Mr Robson had not suggested special treatment, and also in recurrent or even in fulminating cases where the condition was critical. He had only one single experience

of the method, unfortunately not successful. By the courtesy of a colleague it was carried out in a case where a fatal issue was impending and followed twelve hours after operation. At the autopsy an hour-glass contraction was disclosed with the tube projecting slightly into the smaller pyloric sac. The larger cardiac sac had thus remained undrained. The patient had in consequence vomited and this determined a slight fresh bleeding, which was probably aided by the strenuous use of transfusion and stimulants thought necessary.

The lesson learned was that in all cases the tube should be introduced for several inches as far as the lowest point of the fundus, the organ then should be washed out and Cathcart's continuous syphonage established. By such means we may obviate the disagreeable necessity of employing the stomach tube, which is so often badly borne by patients, but which is so indispensable in bringing many cases to a successful termination—gastro-enterostomies even not excepted. At a later date we may utilise the gastrostomy wound for an examination of the interior with the cystoscope. As is well known the gastric fistula after Witzel's operation shortly heals on withdrawal of the tube. The method possesses further the merit that no special or great surgical skill is required for its application.

MR M'GILLIVRAY wished to emphasise the pleasure and benefit he had derived from Mr Mayo Robson's paper. Personally he had had considerable experience in the treatment of hæmorrhage, and he saw many cases recover without operation. He confessed that he did not see how the method proposed by Mr Caird was to have very much benefit on the hæmorrhage. The analogy with the urinary bladder did not seem to hold, as there was the urine always coming in in the latter case that had to be disposed of, and he did not see how a gastrostomy was to give the absolute rest desired. He thought an equal amount of rest might be given by withholding all food.

MR WALLACE had not intended making any remarks, and had only risen because he thought there were two particulars in which the paper was to some extent open to criticism. In the first place hæmatemesis was to his mind only one symptom in connection with affections of the stomach to which it was important that their attention should at the present time be called. They were aware that in recent years various authorities had directed special attention to ulcer of the stomach. Now, bleeding was only one symptom in relation to ulcer, and acute bleeding was, he thought, a rare condition. Excluding carcinoma, he thought great hæmorrhage from ulcer was not common. He had seen a fair amount of bleeding in many cases, but not

many where the physicians, alert as they were now to call in surgical treatment, would consider the hæmorrhage so great as to render this step a necessity.

Then again, there were cases where the hæmorrhage was so great that the patient was very rapidly brought to a condition of *in extremis*. He did not know whether the case referred to by Mr Caird in the students' ward was one which he had seen on several occasions, but he could recall recommending that the stomach should be opened and the stomach and duodenum explored and the vessel ligatured. But this happened several years ago, and the suggestion was not entertained.

He would like to ask Mr Mayo Robson two questions. The first was, whether he was in favour of operation upon a gastric ulcer which was chronic, which had resisted all treatment on the part of the physician for a certain length of time, where the symptoms were not markedly improving and where the patient was running a certain amount of risk of the double condition of perforation and hæmorrhage? Because, although hæmatemesis was an important symptom, it was questionable whether any chronic ulcer of the stomach should be treated medically for a length of time without surgical interference. He was not prepared to enter into the statistics question, but most of them were aware that statistics had been brought forward by one or two, particularly by one of Mickulicz's assistants, in which it was definitely shown that the mortality from gastric ulcer treated solely by the physician was so high as to indicate the necessity for surgical interference; and Mickulicz further points out that the mortality in his own clinique after operation is less than where patients were simply treated by the physician. His second question to Mr Mayo Robson was, what was the percentage of hæmoglobin and what the number of red blood corpuscles in a case that he would consider to be beyond remedial measures? In other words, did he in his operative procedures on gastric cases lay any stress on the blood condition? He thought it was a point of some importance, as it was known, in relation to abdominal surgery in particular, there was great risk of operation because of a marked anæmic condition. The lips might be red and the pulse good, yet these patients might die under operation, without any sepsis or other apparent cause.

DR JAMES felt a certain amount of diffidence in speaking on the question of hæmatemesis, because he was one of those physicians who had never yet had a case in which he considered it necessary to call in the aid of the surgeon. He had looked up some statistics and had got the records of some fifty cases of gastric ulcer which had been under his charge. In these there were 46 with hæmatemesis at one time or another. Of

these 46 several were recurring and very copious. In one case the red blood corpuscles were reduced to some 450,000, and the hæmoglobin to six per cent. But the case recovered. He was delighted to hear what Mr Mayo Robson said as regards treatment; and under the treatment which had been adopted in the cases to which he referred, he had not had a single death in these 50 cases. He would not like for a moment to say that there are no cases in which surgical aid should be called: a case of this sort might arise at any time; and he would say further that he was thoroughly at one with the surgeons in the need for operation where you have a chronic ulcer leading to constriction, or where, as a result of adhesions to surrounding viscera, there was a history of dyspepsia for years. He was then strongly in favour of exploration, and whatever was found necessary in the way of operation.

DR BRAMWELL would mention, firstly, that they were dealing with a subject where all of them were to some extent perhaps taken by surprise. He supposed that they had not been in the habit of thinking very much of the surgical treatment of gastric ulcer, and were very much in the position they were when the surgeons commenced to operate upon the brain. Perhaps their minds were rather predisposed against surgical interference. He was, with Professor Greenfield, much struck with the statistics which Mr Mayo Robson had brought before them, and he hardly thought that modern statistics would at all bear out the conclusions come to. The medical treatment of gastric ulcer had undergone a very marked alteration that had fortunately now made them more successful than when these statistics were published. He referred more particularly to rectal feeding and giving the stomach a complete rest. He was entirely with Mr Mayo Robson in thinking that long rest of the stomach was one of the most important points, but the question then before them was, if they had a case of hæmorrhage from the stomach, was it to be treated by the surgeon or not? In answer to that question, he thought Mr Caird, Dr James, and Dr Affleck had mentioned facts which were of great importance—that the majority of these cases, however severe, got better in the hands of the physician. His own experience was the same as Dr James'. He had never, so far as he remembered at present, seen a fatal case of hæmorrhage from a simple gastric ulcer. He had seen many patients at the point of death, and considered the question of transfusion, but the cases had all ultimately got well. He could not agree with Mr Wallace that copious hæmorrhage from the stomach was an uncommon thing in gastric ulcer. He thought Mr Mayo Robson had answered the question very fairly when he said, let the physician have a fair chance. He would say also, give the patient a much more prolonged period

of rest than has hitherto been done; and then in a small proportion of cases perhaps the surgeon may have to operate. The difficulty was to decide in which cases operation was necessary. He thought it was chiefly in the cases of chronic ulcer with recurrent hæmorrhage that the surgeon should be called in, and they should also remember that all surgeons had not had the experience of Mr Mayo Robson.

MR MAYO ROBSON, in reply, wished that time would permit of his answering all the points that had been brought forward. The discussion had been an extremely interesting one. He thought it had brought out that they practically agreed that some cases should be dealt with by surgical treatment. Most of the speakers argued, and he thought it was quite clear, that the chronic bleeding ulcers could be thus relieved—these, he had no hesitation in saying, could be relieved by a gastro-enterostomy, for instance, which would put the stomach at rest. He was rather astonished to hear Dr Bramwell and others question the statistics which he had quoted. He was not quoting from his own experience. Take Rodman, who had written as lately as 1900, and whose cases are most carefully analysed. He shows 8 per cent. of deaths from hæmorrhage in gastric ulcer. Then Dreschfield, who is a physician pure and simple, and has no special interest in surgery, made the percentage 3½ per cent.; Lebert gives 3 per cent. of deaths, and Steiner gives 6·36 per cent.; Brinton, 5 per cent.; Müller, 11 per cent.; and Welsh, an experienced physician, gives 3·5 per cent. He thought that these authorities were reliable, and that the analyses extended over a very large number of cases, whatever any individual surgeon or physician might say. He did not think they could put these figures aside and say that no deaths do occur, as some physicians would who had spoken. He was not arguing from the experience of one, but of many authorities. He had taken the mean percentage of deaths at from 5 to 7 per cent. Of these from 2½ per cent. to 3 per cent. resulted from acute hæmorrhage, and the other 3 per cent. from chronic hæmorrhage. Now, this 3 per cent. of deaths from recurring hæmorrhage could be saved. Of those due to acute hæmorrhage, he did not say these could be saved. Some of these will die whether treated by physician or surgeon; but what he thought was, that an attempt should be made to save some of these cases. He was extremely interested in Dr James' statistics. They showed that of fifty cases of ulcer forty-six had hæmorrhage. That was very important. He (the speaker) had put down the percentage of cases with hæmorrhage at 50 per cent. to 80 per cent. Dr James' figures amounted to over 90 per cent., and they could be quite certain of their accuracy. It was most

interesting to hear that no deaths had occurred. That surely must have been exceptional. At all events, it is very interesting and important to have something exact to help one to a conclusion. Mr Wallace's questions he thought had been answered in his address, except that referring to the amount of hæmoglobin. He was afraid that he could not answer that; although one ought to be able to give an opinion. He had, however, operated on patients who were as white as the sheets they were lying on, and they had recovered. He was inclined to think it was more a theoretical than a practical matter in acute cases. He was extremely interested in Mr Caird's observations in regard to drainage. He did think there was something in the comparison, although Mr M'Gillivray thought there was not. He thought that a gastro-enterostomy does have the same effect as putting a tube in the bladder; it drains away the contents of the stomach, stops the vermicular movements, prevents hyperacidity, and gives rest. In that way they got the result of morphia administered medically—absolute physiological rest. With regard to Professor Greenfield's question, he had operated on several cases in which a large ulcer, as big as half the palm of one's hand, had eaten through the posterior wall into the pancreas. It was quite hopeless to excise such an ulcer in two of these cases, and a gastro-enterostomy had proved absolutely curative. These two cases were middle-aged men, and are now in perfect health. Just one other remark. He knew that cases of gastric ulcer were treated properly while under medical charge. But, as a matter of fact, cases of chronic ulcer did go about; he was seeing them frequently—they went about month after month, and year after year—some had been operated on after a duration of twenty-five years. He wished he could answer all the questions put, but unfortunately trains would not wait. He had been very glad to hear Sir William Gairdner's concession to the treatment he had advocated. He hoped it would be seen that he did not propose that surgeons should take over all these cases; he wanted to be quite clear that surgeons should take over no cases apart from the physician. All these cases should have medical treatment first, and then surgical treatment should follow. He thanked them all for their very kind reception.

Meeting VI.—March 6th, 1901

MR A. GORDON MILLER, *President, in the Chair*

The President reported that the following address had been forwarded for presentation to His Majesty King Edward VII.

Unto the King's Most Excellent Majesty, the Loyal Address of the Medico-Chirurgical Society of Edinburgh.

May it please Your Majesty, We, Your Majesty's dutiful and loyal subjects, the President, and members of the Edinburgh Medico-Chirurgical Society, humbly approach Your Majesty with the assurance of our profound sympathy in the great loss which Your Majesty and the whole nation have sustained in the death of Your Majesty's August Mother, our late beloved and revered Sovereign. We recall with feelings of sincere gratitude the sagacity and constant care which Her Majesty brought to bear throughout the many years of her illustrious reign upon all matters which tended to the promotion of her subjects' welfare and happiness.

We tender to Your Majesty our most respectful congratulations upon Your Majesty's accession to the Throne. We assure you of our most dutiful and loyal allegiance, and we pray that Your Majesty may long be spared to rule over a happy and contented people.

(Signed) A. G. MILLER, F.R.C.S.E.
President.

EDINBURGH, *Feb. 8th*, 1901.

I. ELECTION OF MEMBERS

George Lyall Chiene, M.B., C.M., 26 Charlotte Square, was elected a member.

II. EXHIBITION OF SPECIMENS

1. *Dr A. Bruce* for *Dr J. O. Affleck* exhibited a greatly DILATED HEART, from a woman, aged 69, who had been a patient in the Longmore Hospital for seven years. The heart was uniformly dilated. There was great triscupid regurgitation and the veins of the neck were enormously enlarged. The liver, too, was much enlarged, and gave very distinctly the sign of hepatic pulsation. There was no albuminuria, and no general dropsy; but ascites was present from the time of the patient's admission until her death. The abdomen was tapped 188

times and 54,494 ounces of fluid had been removed. The largest amount of fluid removed at one time was 414 ounces.

2. *Mr Alexis Thomson* exhibited specimen of MULTIPLE CARTILAGINOUS LOOSE BODIES FROM THE KNEE-JOINT.

The patient was a woman, aged 36, who had suffered from articular rheumatism at the ages of 12 and 16 years. At the age of 17, the left knee became gradually swollen and painful; it did not prevent her doing active work on a farm. At the age of 27 she noticed the presence of loose bodies in the joint. The swelling gradually increased until about eight months ago, when she became lame and could only walk slowly and with difficulty. There was complaint of pains and stiffness, especially after sitting, and the pain interfered with sleep. Dr Robertson drew off three ounces clear fluid, with considerable relief to the symptoms. When seen by Mr Thomson, there was great swelling of the knee, mainly involving the upper pouch; this was filled with hard uneven loose bodies which could be made to move up on one another like dried peas in a bag; there was a moderate amount of fluid in the joint. The quadriceps extensor was slightly wasted. The range of movement was very limited and the joint could not be completely extended. The general health was good and the other joints appeared to be quite normal.

The upper pouch of the knee-joint was incised on the outer aspect; the synovial membrane was very vascular and its free surface was covered with delicate thread-like processes; a portion was removed for microscopical examination. About six ounces of clear amber-coloured mucin containing fluid escaped. The loose bodies were shelled out with difficulty as they tended to cake together into large masses and to adhere to the synovial membrane. The articular surfaces appeared to be healthy. The wound was stitched up without drainage.

The bodies numbered several hundred and varied in size from a pepper grain to a small hazel nut; they are extremely irregular in shape, being made up of rounded nodules; they are pearly white in colour and of the consistence of cartilage. Microscopical examination showed them to consist of hyaline cartilage with scattered areas of calcification. The synovial membrane showed no trace of any recognized disease; its fine surface was studded with villous processes consisting of large

capillary blood-vessels surrounded by young connective tissue corpuscles.

Mr Thomson did not regard the loose bodies as originating from any of the known types of joint disease, but as being of the nature of new growths or tumours in the synovial membrane and that the changes in the latter, as above described, were secondary to the loose bodies, being the result of their mechanical pressure and irritation.

3. *Dr Dawson Turner* exhibited Röntgen photograph of RENAL CALCULI from a man, P.C., æt. 50.

History.—Passed a small stone "per urethram" eleven years ago, after suffering from the usual symptoms of the passage of a stone from the kidney (right). Shortly after he passed another stone with same symptoms from left kidney. Since then he has constantly suffered pain in both kidneys, but principally in right, with recurrent bleedings, especially when he made extra exertion. On admission, there was marked enlargement of right kidney with tenderness on pressure. Dr Dawson Turner made a very successful skiagraph, which showed three distinct shadows in the right kidney and a less apparent one shadow in the left kidney.

Operation.—Right kidney exposed, found to be hydronephrotic and contained three calculi which were removed. Left kidney not interfered with. Patient passed urine after operation, but early next morning a severe hæmaturesis took place and continued until he died.

P.M.—No stones remained in right kidney, but left kidney contained a large number of calculi.

III. DEMONSTRATION [1]

Dr Chalmers Watson gave a histological demonstration on THE COMPARATIVE PATHOLOGY OF ATAXIA; THE PRIMARY IMPORTANCE OF VASCULAR DISEASE.

The special object of this demonstration, and also of the former one described on page 62, was to show that the vascular changes in the cord were the primary factor and the involvement of the nerve fibres a secondary occurrence in disease in the horse which appeared to bear a real relationship to

[1] The original work comprised in this demonstration was carried out in the Laboratory of the Scottish Asylums, where the author received valuable assistance from the Director, Dr Ford Robertson. He is also much indebted to Mrs Chalmers Watson, M.D., for valued assistance in the investigation.

FIG. I.—The stifle joints, suspended by the lower end of the femur. The illustration gives a general view of the great enlargement of the soft tissues, and shows two bony masses found encapsulated *outside* the joint. The fluid in the joints had been previously lost.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

FIG. II.—The interior of the stifle joint. The anterior part of the capsule, with the patella, has been reflected downwards and forwards.

NOTE—(*a*) The marked enlargement of the soft tissues. The thickness was 1¼ inches at the position of the broad white line.
(*b*) The enlargement of the lower end of the femur. The illustration gives a faint indication of the irregular thickening of bone at the upper margin; a similar irregularity is present at one margin of the patella.
(*c*) An area of ulceration is visible on the articular surface of the patella.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

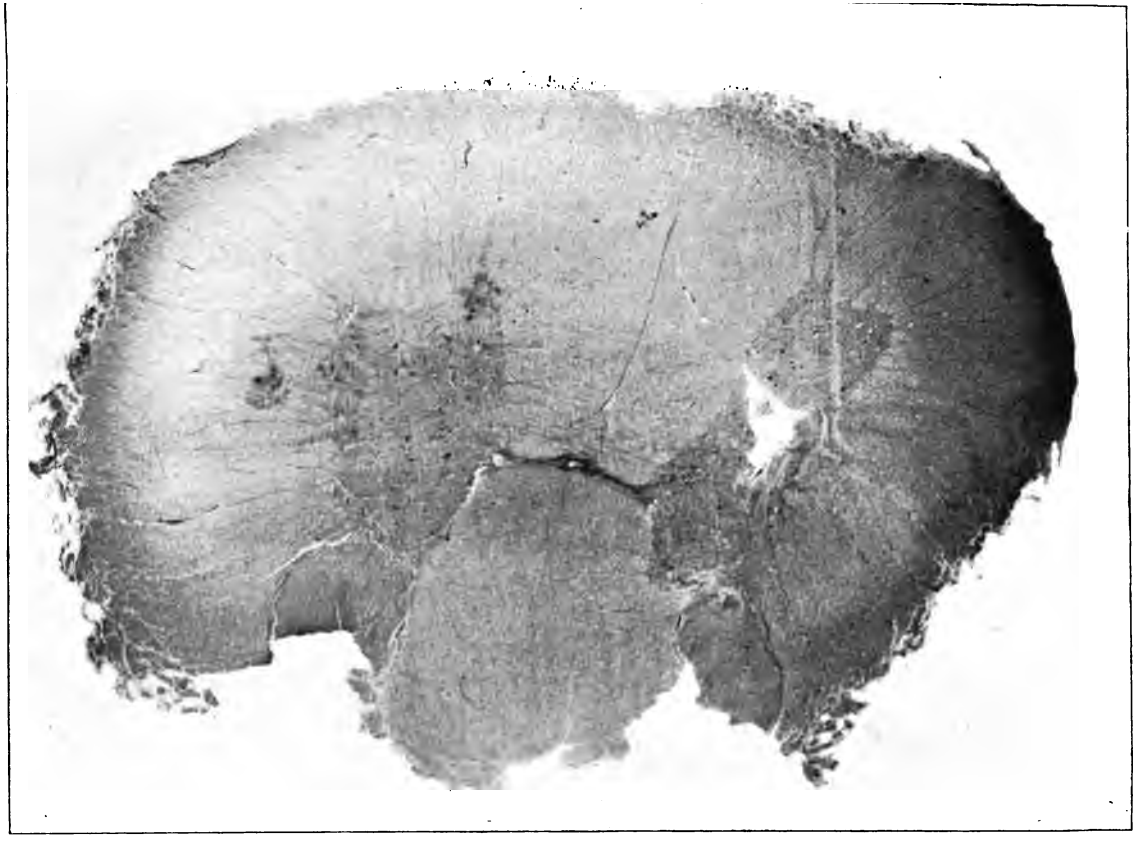


FIG. III.—Lumbar cord. Photoxylin section stained with hæmatoxylin and eosin. Shows—(*a*) Mark loss of fibres in the posterior columns and in the region of the posterior nerve roots. (*b*) Cavity in the anterior cornu. (*c*) The irregularity visible at the margin of the cord anteriorly is due to the rarefaction of nerve fibres referred to in the text (*q.v.*) *See also Fig. IV.* × 10.

NOTE.—The differences in the colour of the various columns in this illustration are misleading. For an account of their condition see text.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

FIG. IV.—Shows one of the many diseased vessels at the margin of the softened area in the posterior columns of the lumbar cord. The change consists in advanced hyaline and fibroid thickening, which has probably led to complete obliteration. × 200.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

FIG. V.—Dorsal cord. Marchi method. Note the dark stained area in the posterior columns, an ascending degeneration due to the destruction of the nerve fibres in the lower part of the cord. The illustration does not indicate the scattered degenerated fibres in the rest of the cord. × 10.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

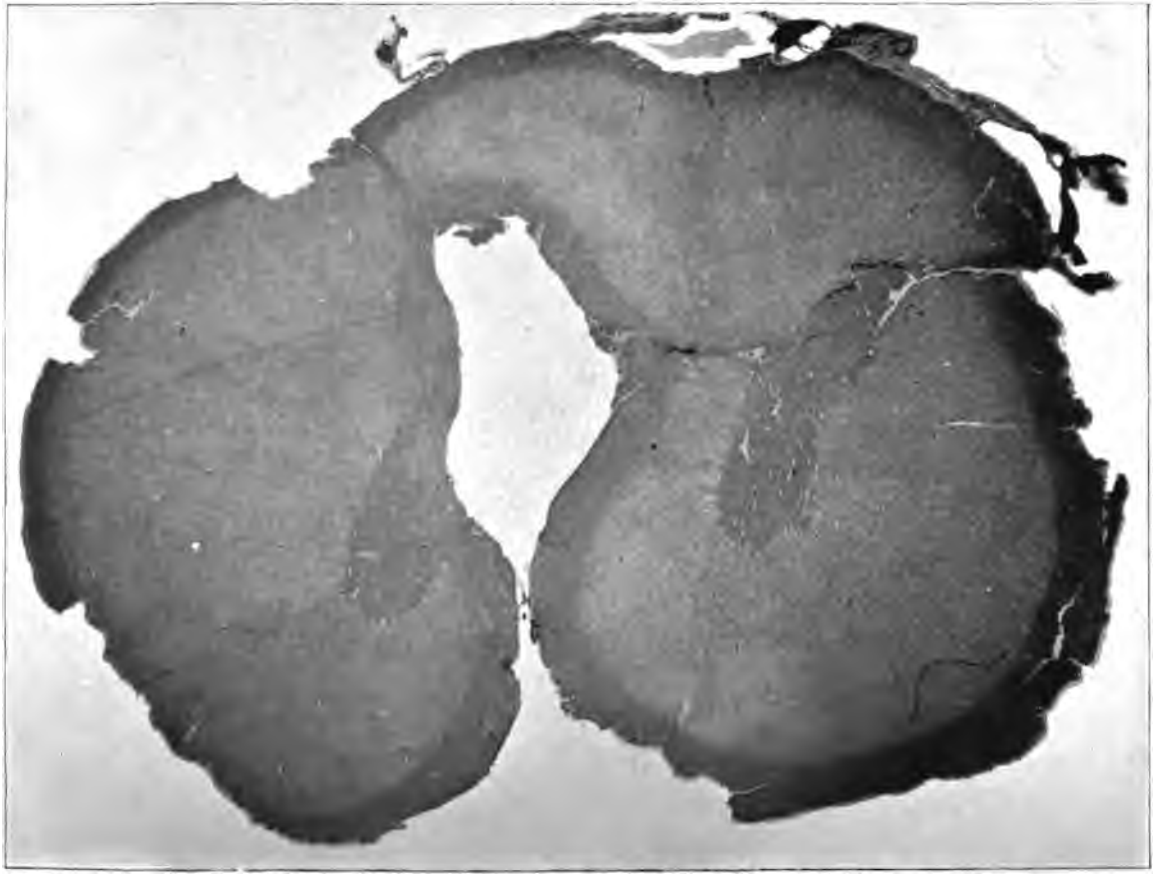


FIG. VI.—Cervical cord. Photoxylin section stained with hæmatoxylin and eosin. Shows a very large cavity (syringomyelia) involving a large part of the grey matter on one side, and extending right out to the periphery posteriorly. The black dots and dark line in the neck of the cavity are diseased vessels. *See Fig. VII.* × 10.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

FIG. VII.—Higher power view of the vessels referred to in Fig. VI. It shows very advanced fibroid and hyaline degenerations of these vessels,—one in longitudinal and two in transverse sections. The former is apparently the vessel whose obliteration has been followed by the cavity formation. × 170.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

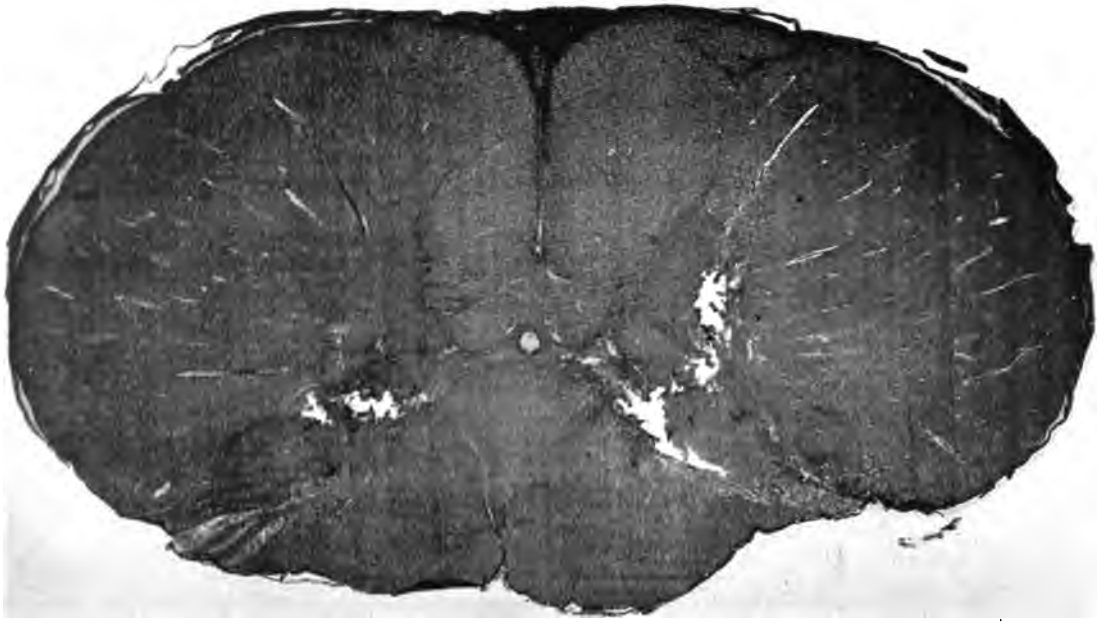


FIG. VIII.—Cervical cord. First cervical. Photoxylin section. Hæmatoxylin and eosin. (× 10.)
Note the irregular cavity formation in the grey matter; also the marked depression on the surface at the exit of one posterior nerve root, the result of a previous lesion.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

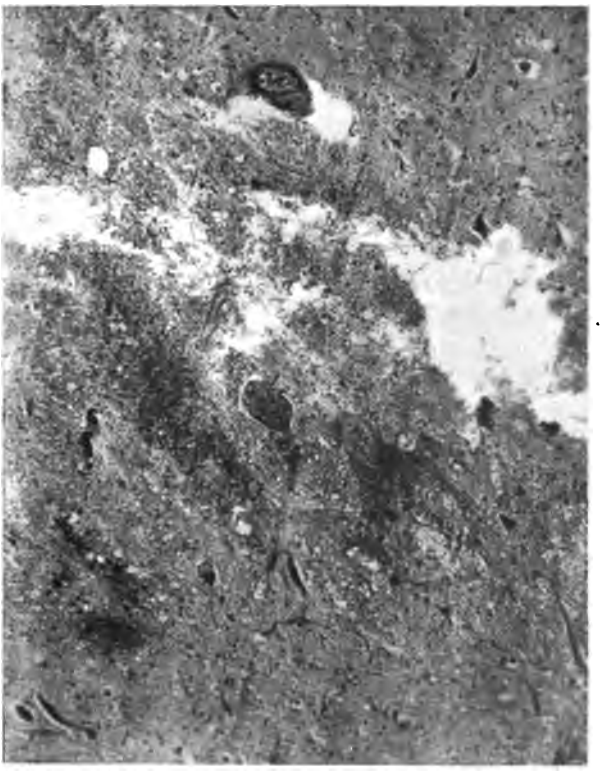


FIG. IX.—High power view of grey matter in Fig. I. (× 100.) The dark areas are recent hæmorrhages, which are numerous, and vary in size as shown.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

FIG. X.—Cervical cord. Photoxylin section. Hæmatoxylin and eosin. (× 55.)
The periphery of one lateral column in which there is considerable degeneration of nerve fibres.

NOTE—(i.) The pronounced thickening of the vessels.
(ii.) The dilated perivascular lymphatic spaces scattered throughout the field. Note the moniliform arrangement in the near part of the field.
(iii.) The slight increase of the pia.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

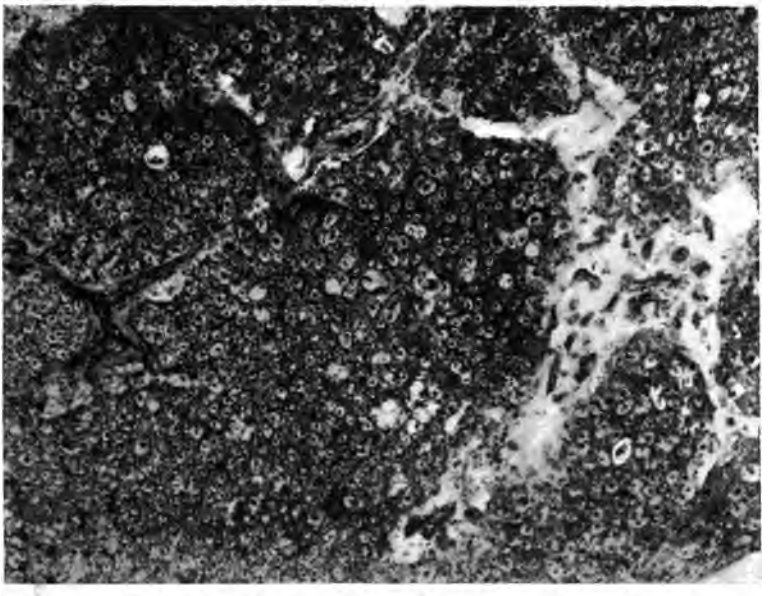


FIG. XI.—Posterior column of same section as Fig. III.
Note the irregular area of atrophic softening in which the remains of markedly thickened vessels are visible. To the left are seen vessels showing pronounced fibroid changes.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

FIG. XII.—Cervical cord. Weigert stain. (× 6.)
Shows extensive degeneration of the nerve fibres, most marked in the posterior columns, and anteriorly along both sides of the septum. It is present, though in less degree, in the lateral columns, especially at the margin of the cord.

(These appearances are best seen if the illustration be held at a distance of two feet.)

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

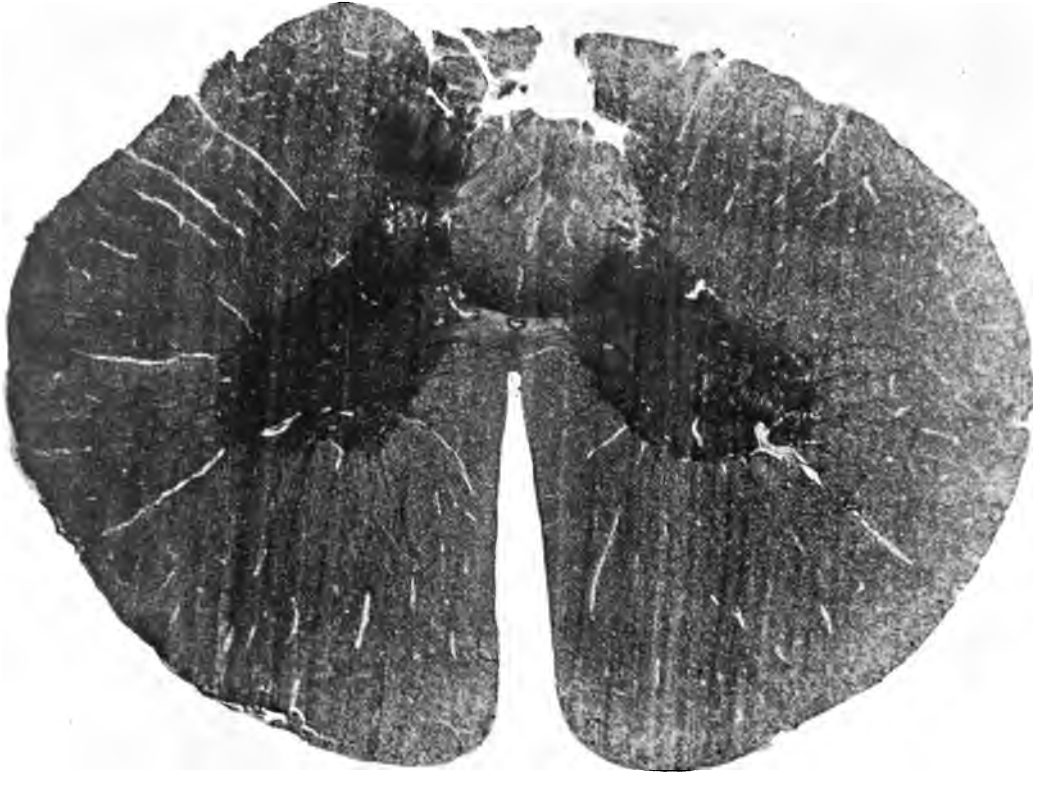


FIG. XIII.—Lumbar cord. Photoxylin section. Hæmatoxylin and eosin. (× 8.)
Shows an area of acute softening in the superficial part of the posterior columns.

TO ILLUSTRATE DR CHALMERS WATSON'S PAPER.

tabes in the human subject. The records here appended and elsewhere given in detail (1, 2) are the first attempts at a systematic clinical and pathological description of these affections in the lower animals, and the author drew attention to the importance of this line of investigation as one of great value for elucidating some important problems of neural pathology. He emphasised two points. In the first place, and in opposition to some who hold opposing views, he stated that it is of the very greatest fundamental importance to determine whether the toxic agent in these affections acts primarily on the nerve cells and nerve fibres, or on the vessels which supply them, and in his opinion the views almost universally held in this country at the present time as to the neural degeneration being primary and the vascular changes secondary, are erroneous. And secondly, he laid emphasis on the fact that this view by no means excludes the consideration of a varying vitality of neurons as an important factor in these diseases. This statement is necessary as we frequently find that the advocates of the neural theory unwarrantably assume that those who support the vascular theory of the disease neglect to take the state of the nerve cells and fibres into due consideration.

With regard to the illustrations, it should be stated that all tissues which were unduly soft in consistence or contained small or large cavities were fixed in photoxylin, so as to avoid the risk of artifacts, and none of the cavities shown can reasonably be regarded as artificial. A short description of the subject which furnished the illustrations Figs. 1 to 7 is given on page 62, and a complete record of the case will be found in veterinary literature.

The second demonstration was taken from the case of a hackney, 18 months old, which exhibited cord symptoms similar to those recorded of the previous case, which led the author to diagnose a lesion of the cord, most marked in the posterior part, of a nature akin to Friedreich's ataxia in the human subject. The diagnosis was confirmed at the post mortem examination. The animal had "wintered badly," probably the result of six months' injudicious dietary. As in the former case, the special object of the demonstration was to illustrate the primary importance of vascular disease in the production of the pathological and clinical picture. (*Vide* Figs. 8 to 13.) The histological appearances supported the author's

views :—(1) That vascular disease in the cord plays a much more important *rôle* than is commonly believed ; (2) that the lesions in the cord are the local expressions of a general disorder rather than distinct entities of "nervous" origin ; (3) that they are manifestations of chronic autointoxication from a general disorder of nutrition, with the alimentary canal as the principal focus; (4) that these conditions ought to be curable in their early stages. Confirmatory evidence from human pathology was shown, as follows: (*a*) A section of the lumbar cord from a case of tabes of thirteen months' duration, to show softening and degeneration of fibres in relation to advanced vascular disease (the patient died from malignant disease). The results of this examination confirmed other pathological records of early cases of tabes, which showed that the primary change was the vascular one. [As tabes is not a fatal disease, these records are necessarily exceedingly rare.] The author was indebted to Dr Fleming, Pathologist to the Royal Infirmary, for the piece of cord submitted to investigation. (*b*) Section of the cord from a case of long standing hemiplegia to show that the degeneration of the fibres of the crossed pyramidal tract was not associated with a marked vascular lesion. The author was indebted to Dr James for this tissue.

A complete record of these cases, with a consideration of their relationship to allied affections in the human subject, will be found in the author's papers, viz. :—

1. A contribution to our knowledge of disease of the nervous system in horses.—*The Veterinary Journal*, March 1901.
2. On diseases in the nervous system of horses : Hereditary Ataxia.—*The Veterinary Journal*, June 1901.
3. Pathogenesis of tabes and allied condition in the cord.—*Brit. Med. Journal*, June 1, 1901.

V. Original Communications

1. STENOSIS OF THE SMALL INTESTINE AFTER STRANGULATED HERNIA

By Alexis Thomson, F.R.C.S.Ed., Lecturer on Surgery, and Assistant Surgeon, Royal Infirmary, Edinburgh

It is remarkable how completely the bowel recovers from the damage sustained in a strangulated hernia. It is almost the invariable experience that unless gangrene has occurred, the

bowel may be returned into the abdomen with every confidence that it will recover. We need not be concerned because of the existence of recent peritonitis and the presence of lymph on the bowel as it is quite exceptional for this to result in the formation of adhesions and bands which may cause obstruction by kinking or strangulation.

The object of this communication is to direct attention to the mechanical constriction of the bowel in strangulated hernia apart from its connection with gangrene and perforation.

In operating for strangulated hernia it is very common to meet with circular grooving of the bowel at one or both ends of the loop engaged in the hernia. These constriction grooves or furrows may be shallow or deep, may occupy only a segment of the circumference or extend right round the bowel. When the intestine is liberated and handled, the grooves almost invariably recover. They may, however, persist; in this case, the symptoms of obstruction continue instead of being relieved by the operation; or at a later period they may become the seat of a fibrous stricture; in this case the patient appears to be making a good recovery until the symptoms of obstruction gradually reassert themselves.

1. The stenosis which results from persistance of the constriction grooves is an urgent condition. An apparently successful herniotomy is not followed by relief of the symptoms. The patient continues to vomit, loses strength rapidly, and if left to himself will almost inevitably die. If, however, the belly is opened under these conditions, we find a state of matters which is quite capable of being relieved. The damaged loop of bowel is found in the vicinity of the hernial opening; although two or three days may have elapsed since the herniotomy, the intestine has not altered either in the direction of recovery or of gangrene. It presents the same discoloration and the same constriction grooves as when seen at the first operation. The obstruction resulting from this persistance of the constriction grooves may be rendered more complete by the addition of paresis of the muscular coat of the gut; in our opinion, however, the mechanical constriction is the primary and essential factor.

The microscopical appearances of the bowel at the seat of constriction consist in great engorgement of the vessels and extensive and hæmorrhagic and leucocytic infiltration of all the

coats, being most pronounced in the mucous membrane; similar changes are present in a minor degree in the majority of cases of strangulated hernia.

The treatment consists either in the immediate resection of the damaged portion of bowel or in relieving the obstruction by enterotomy and deferring the resection until the associated conditions are likely to favour its successful performance. I am strongly in favour of the latter alternative.

I shall now relate an illustrative case which occurred in my private practice a little more than a year ago.

CASE I.—The patient was a male child, æt. 2. He was known to be the subject of imperfectly descended testicle and inguinal hernia on both sides. In September 1899, the right hernia became strangulated but was reduced under chloroform. In February 1900, he was again seized with sudden illness, cried with pain, vomited, and the left hernia was found to be strangulated.

It was impossible to reduce it under chloroform. Dr Robert Stirling, of Perth, was called to see the child, and performed herniotomy within six hours of the onset of symptoms. The sac contained a loop of small intestine, shining, quite black, and with a well marked constriction at either end of the loop where it had been caught at the internal ring. The bowel was regarded as viable, and was returned within the belly. The testicle was left in the inguinal canal. The symptoms of obstruction persisted. Dr Robert Stirling, being unfortunately confined to the house through illness, I was summoned to see the child three days after the herniotomy. I found him exhausted and reduced with the constant vomiting and want of nourishment. The pulse was 120. I believed the obstruction to depend upon some complication related to the recent strangulation, and advised immediate operation. An interesting feature was the entire absence of abdominal distension, doubtless to be attributed to the obstruction being situated very high up in the jejunum; as a matter of fact it was found within two feet of the duodeno-jejunal junction.

The rectum being full of fæces was emptied by means of an enema. Mr Robert Purves helped me with the operation. The wound was extended upwards so as to open the belly. A loop of bowel, quite black in colour, was at once exposed, showing a deep constriction ring at either end of the loop as

if a string had been tied round it. The enormously distended bowel alone was traced beneath the transverse colon to the duodenal junction; below, it was empty and contracted to the size of an ordinary lead-pencil. The mesentery of the damaged portion was also black.

The critical condition of the child did not permit of an immediate enterectomy; the damaged loop and distended portion above were brought out and fixed in the wound, and a Paul's tube was inserted into the distended bowel. Ten days later, the wound was again opened up and the damaged loop of bowel was removed along with its mesentery. In proceeding to join the ends it was found that the lower contracted end was only about half the diameter of the upper; it was therefore cut very obliquely, and a longitudinal incision was made on the side opposite the mesenteric attachment; the junction was effected by a double row of sutures. His recovery was delayed by an attack of bronchitis, which led to the formation of a ventral hernia at the upper end of the scar. The hernia was cured with a truss and the child is now strong and well.

The portion of bowel removed measured 23 cm.; its proximal end had a diameter of 2 cm., the distal end of 5 mm. Sections were made in the situation of the distal constriction; there was no appearance of necrosis of the mucosa, merely extensive hæmorrhagic and leucocytic infiltration of all the coats and especially of the mucous membrane.

2. The stenosis, which depends on the formation of a fibrous stricture, is of gradual development and the associated symptoms do not make their appearance until an interval of weeks has elapsed since the original strangulation. The herniotomy is apparently successful, the vomiting ceases and the bowels act naturally, or there may be persistent diarrhœa. After an interval averaging about three to five weeks, but which may extend to several months or years, the patient complains of pain in the belly, especially after solid food, and he occasionally vomits after meals. The symptoms are relieved for a time by laxatives and a restricted fluid diet, but obstruction gradually develops and becomes more and more decided. Where the stricture is complicated with adhesions and kinking of the bowel, the onset of obstruction is more rapid and sometimes attains the urgency of the acute variety. The second operation has been performed at periods varying from the

sixteenth day to the twelfth week after the original herniotomy. When the abdomen is opened, the damaged loop of intestine is usually found in the vicinity of the previous wound and is frequently discovered to be fixed in this region by adhesions to the belly wall or to the neighbouring organs. The adhesions having been separated, the affected loop is found to present a fibrous stricture at one or at both ends, in the position of the constriction rings observed at the first operation. The mesentery may be contracted and fibrous. Above the stricture the intestine is greatly distended and its walls hypertrophied while below it is collapsed and empty.

Under these conditions the treatment is directed towards relieving the obstruction and getting rid of the stricture. If there is no great disproportion between the bowel above and below, and the patient's general condition is favourable, the strictured loop may be resected straight away. Personally, I am in favour of relieving the obstruction in the first instance by means of an enterotomy above the proximal stricture, and resecting the bowel at a later period when the distension has been got rid off. Enteroplasty or longitudinal division of the stricture, and the exclusion of the strictured loop, by making an anastomosis between the bowel above and below, are alternative, but less desirable procedures.

The nature of the stricture varies; it may be a mere cicatricial furrowing of the peritoneal coat, an annular or tubular fibrous stricture involving the entire thickness of the bowel or a septum or diaphragm crossing the bowel and provided with an opening in its centre. Necrosis and shedding of the mucosa is regarded as the most common antecedent of the fibrous stricture.

The following is a characteristic example of stricture of the bowel resulting from strangulated hernia; as it has already been reported elsewhere[1] in full, I shall only recapitulate its more important features.

CASE II.—In November 1896, I operated upon a man, aet. 31, for congenital inguinal hernia on the left side, which had been strangulated for three days. The sac and its contents—a loop of intestine—presented the usual features of recent peritonitis. Having relieved the light constriction at the internal ring, the bowel was pulled down and was found to

[1] *Brit. Med. Jour.*, October 10, 1897.

present two constriction rings, one at either end of the loop, in the position of the internal ring. It was regarded as viable and returned. The patient's progress was quite satisfactory until the end of the third week, when he began to present symptoms of chronic intestinal obstruction. Laparotomy was recommended, but was not accepted until twelve weeks after the original herniotomy. He was then emaciated to a skeleton, the belly was distended with visible coils of small intestine arranged in a ladder-pattern and exhibiting active penstaltic movements attended with loud gurgling noises. He vomited large quantities of foul smelling greenish brown fluid every alternate day.

The belly was opened above and to the inner side of the existing scar. The loop of damaged bowel was found close to the inner edge of the wound, being fixed by an adhesion to the anterior abdominal wall. It was liberated and brought outside the wound. There was a well-marked fibrous stricture at either end of the loop; above the stricture the bowel was distended and hypertrophied while below it was contracted and empty. Enterotomy was performed in the dilated portion, Sixteen days later, the damaged bowel was resected and the ends were joined by means of a Murphy button. He was up and out of bed in a fortnight and passed the button on the fifty-first day. He has been in the best of health ever since.

The portion of bowel removed measured 43 cm. The distended upper end possessed a lumen of 5 cm. in diameter. The proximal stricture was 1·5 cm. in length and 8 mm. in diameter and its wall consisted of firm fibrous tissue; microscopical examination showed that the mucous and submucous coats had been entirely replaced by fibrous tissue. The distal stricture took the form of a shallow circular groove. resulting from the formation of fibrous tissue in the situation of the peritoneum and outer longitudinal muscular coat.

Similar cases of stricture of the gut following strangulated inguinal hernia have been recorded by Nicaise, Garré, Maas, Kellock and others. Mr Caird has described and figured a narrow pyloric-like contraction which developed at the seat of constriction after operation for strangulated femoral hernia, and which (operative interference being refused) proved fatal seven months later.

2. MYASTHENIA GRAVIS[1]

By EDWIN BRAMWELL, M.B., M.R.C.P., Lond. and Edin., Edinburgh

THE purpose of this paper is to draw attention to a disease which is as yet little known to the profession, no case of myasthenia gravis having been, so far as I know, yet recorded in Scotland. Although certainly a rare disease, I venture to think that myasthenia gravis is a condition with which every practitioner should be acquainted, since it is often mistaken for hysteria, since it is often fatal, and since in some cases, at least, a fatal termination can be averted by appropriate treatment. I have been fortunate in having had the opportunity of examining nine cases of the disease, a larger number probably than has come under the notice of anyone in this country. Up to the present time only some eighty or possibly ninety cases have been reported.

In this communication I propose to describe briefly the more important clinical features of the disease. The following case, which was kindly sent to me by Dr Miller of Dundee, is typical:—

An unmarried female, aged 23, a school teacher, was seen on 10th November 1900. She complained of difficulty in speaking and of weakness in the hands and arms. Excepting that her mother was a somewhat nervous woman, the family history was excellent. Three years previously the patient had suffered for a few months from bloodlessness, otherwise she had enjoyed good health up to the commencement of the present illness. The first symptom, viz., difficulty in speaking, especially after talking for some time, was noticed ten weeks ago on her return from a two months' holiday. During the following six weeks the difficulty in speaking became worse, and it was noticed that she spoke through her nose—when she rose from bed in the morning speech was always natural and easy, and it was not until the day was well advanced that she experienced any inconvenience. The lips used to become stiff and un-

[1] Many cases of this disease have been described as cases of asthenic bulbar palsy. This nomenclature is, I think, most misleading, and should be discontinued for the following reasons:—Firstly, the symptoms are not limited to the bulbar muscles; it is indeed quite exceptional for the limbs and trunk to escape; and secondly, from what is known of the disease I think it probable that cases occur in which the extremities are alone affected, in the earlier stages of the disease at least. A case of this kind is recorded by Ormerod in the *St Bartholomew's Hospital Reports*, vol. xxxi. p. 50.

manageable, and her articulation was consequently defective. At night saliva ran from her mouth on to the pillow. When she drank, fluids regurgitated through the nose. The jaws became easily tired when eating, and there was difficulty in swallowing solids. The neck felt weak, and she had difficulty in supporting her head. When brushing her hair at night her arms became easily tired, and she was obliged to rest them repeatedly. After playing on the piano for a short time her hands speedily became fatigued, so that she was unable to stretch an octave. She had had several attacks of breathlessness. All these symptoms were more pronounced towards evening and if she was excited. In the morning she usually felt perfectly well. Not only was any muscular exertion followed by fatigue in the muscles involved, but as she herself expressed it, "The using of any muscles seems to have that effect on the others." For a fortnight before I saw her she had been resting from work, and her symptoms had improved considerably.

The patient's state on 10th November 1900 was as follows:—

A well-developed, well-nourished, and intelligent girl; she is not anæmic; she answers questions promptly; her memory is good and she does not appear to be emotional. There is a lack of expression about her face, and when she speaks a want of mobility of the lips. There is no facial asymmetry, and she can make satisfactorily such movements as frowning, whistling, and showing the teeth. She cannot, however, wrinkle her forehead; when she is told to close her eyes tightly, little force is required to open them. There is no distinct weakness of the oral sphincter. Her speech is a little indistinct, but the defect is trifling; when, however, she reads aloud from a book, articulation becomes gradually more indistinct, her utterance nasal and the voice weaker, while she has to stop frequently for breath. There is, in fact, a combination of defective articulation and phonation. There is no ptosis, nor is there any tendency for the lids to drop when she continues looking upwards for any length of time. The eyes are not prominent. The ocular movements are perfect; there is no nystagmus. The pupils are equal, of moderate size, and react perfectly both to light and on accommodation. No weakness in the muscles of mastication is to be detected. Although she swallows slowly,

there is no noticeable defect in the act of deglutition. The palate movement is impaired and its reflex irritability is diminished, especially on the left side.

Dr Logan Turner, who kindly examined the larynx, was of opinion that the cords came together well and that abduction was perfect.

The tongue is protruded straight and its movements are satisfactory.

The special senses are unaffected. The acuity of sight and hearing is good, there is no diplopia and the discs are normal. No defect of the visual fields is to be detected by the rough test.

With the exception of doubtful weakness of the muscles of the back of the neck, the movements of the limbs and trunk are good. She is unable, however, to hold the arms above the head for more than half a minute. She has noticed no weakness in the legs and can walk four or five miles without feeling tired. Her gait is quite natural. There is no muscular wasting and no fibrillary twitchings are to be seen. Although an exhaustive electrical examination was not made, the myasthenic reaction was quite evident in the right biceps muscle.

Excepting for a sense of tiredness, amounting to pain, in the muscles of the neck and in the arms after they have been much used, there are no subjective sensory disturbances. Of late she has not been sleeping so well as usual; she sometimes feels too tired to sleep.

A careful examination revealed no objective sensory defect.

As to the reflexes. There is a brisk jaw jerk. The arm jerks are active, those on the left side being distinctly greater than on the right. The knee and Achilles jerks are well marked. There is no ankle clonus. The plantar reflex is of the flexor type. There has been no affection of micturition; her bowels are regular.

Her appetite and digestion are good. The heart is of normal size, the sounds pure, the pulse regular and about 90 per minute. She had had no attacks of palpitation.

She has a slight feeble cough, and suffers considerable inconvenience from inability to cough up the phlegm which collects in the pharynx in consequence of a pharyngitis from which she is at present suffering.

Menstruation is regular and natural. There is no enlargement of the thyroid gland. The urine was not examined.

Let me in a few sentences epitomise the clinical history of this case.

A girl of twenty-three, who had previously enjoyed good health, without apparent exciting cause gradually develops difficulty in speaking, swallowing, and chewing, weakness of the ocular muscles, of the palate, of the neck, and of the arms. In the morning she is usually free from symptoms, but as the day goes on, weakness in these various muscles develops. If she continues using any of the above-mentioned muscles, a state of temporary paresis results, associated with a feeling of fatigue. The muscular weakness is unaccompanied by atrophy; the faradic contractibility in the affected muscles (biceps) is rapidly exhausted; the deep reflexes are brisk; the plantar reflex is of the flexor (normal) type; the sphincters are unaffected; there is no objective disturbance of sensation, and no affection of the special senses.

Symptomatology.—The most prominent clinical feature of myasthenia gravis is the facility with which the muscles become fatigued by voluntary effort. The term *myasthenic state* seems applicable to this phenomenon. A voluntary movement, which is at first perfectly carried out, becomes rapidly feebler each time it is repeated; finally, if persisted in, *all* power of performing the movement may be temporarily lost; after a short rest the movement can be again performed with almost as much power as on the first attempt. A variable degree of persistent paresis is often present in the affected muscles. This paresis is usually slight, but may amount to a complete paralysis (*e.g.* complete ophthalmoplegia externa); it probably represents a more advanced stage of the myasthenic state, in which the recuperative power of the neuro-motor apparatus is impaired in an unusual degree.

Just as varying degrees of motor weakness are met with, so corresponding variations in the Faradic excitability of the muscles occur. It is, however, the rapidity with which the muscles become fatigued by the Faradic current which is especially characteristic of this disease; the term *myasthenic reaction* has been given to this condition. If a tetanising current be applied to an affected muscle, at first an active contraction is obtained, but in the course of a short time the muscle ceases to respond. The myasthenic reaction is not constantly present; at one time a muscle can be exhausted

in half a minute, at another time it may be impossible to exhaust it at all. The myasthenic reaction, although not absolutely pathognomonic of the malady, is a diagnostic sign of great value.

The muscles react normally to galvanism.

Pathology.—The scope of this paper will only allow of brief reference to the pathology of the disease. Of eighteen cases collected and analysed by Harry Campbell and myself which had been examined *post-mortem*, in none was there a lesion which would satisfactorily account for the symptoms present during life. Dejerine and Thomas, it is true, at the Paris Congress of last year reported definite "findings" in a case of myasthenia gravis, but it remains to be seen whether future observations will show that these changes are constant and are the cause of the symptoms.

There is reason for supposing that the disease is due to a toxin which is, I think, probably of endogenous origin. Possibly, too, in the subjects of myasthenia gravis there may be some congenital defect or abnormality either in the construction or mode of functionating of the neuro-motor apparatus.

The majority of writers believe that the seat of the lesion is in the motor nervous system rather than in the muscles, and clinical evidence suggests that it is the lower motor neuron which chiefly suffers. Males and females are both liable to the disease, which occurs most frequently between the ages of twenty and forty. Some cases have followed upon an acute infective disease, but it seems probable, I think, that in these cases the infective disease acts, not as the primary exciting cause but rather as a predisposing agent, by lowering the resistance of the tissues. In about a quarter of the recorded cases there has been a neuropathic heredity.

Diagnosis.—Turning to the question of diagnosis. Where the symptoms are well marked the diagnosis of myasthenia gravis is easy. The recognition of slight, and especially of complicated cases, may, on the other hand, be a matter of great difficulty. The following are the chief points upon which the diagnosis is to be based :—

Firstly.—The presence of weakness, which is often slight in degree and is unassociated with atrophy—the muscular weakness varies greatly in intensity from time to time and tends to

affect in varying degree all the voluntary muscles—the muscles of the extremities and trunk as well as those innervated by the cranial nerves.

Secondly.—The facility with which the muscles become exhausted both by voluntary effort (myasthenic state) and by Faradism (myasthenic reaction).

Thirdly.—The absence of sensory symptoms, apart from the fatigue of exhaustion, of sphincter trouble, and of mental disturbance. The deep reflexes are usually brisk, while the plantar reflex shows no characteristic alteration from the normal.

In differentiating myasthenia gravis from hysteria the absence of a neurotic temperament and of sensory symptoms and the presence of ocular and facial paresis, weakness of the neck muscles and of the myasthenic reaction are the chief points of diagnostic value.

More advanced cases of myasthenia gravis are often mistaken for chronic progressive bulbar palsy.

Points of special diagnostic importance are the absence of muscular atrophy in myasthenia, the presence of ocular and upper facial paresis, of the myasthenic reaction, and, above all, of variability in the severity of the symptoms.

Prognosis.—The prognosis in any given case of myasthenia gravis is a matter of great uncertainty. In most cases the disease runs a course which is characterised by temporary improvement and relapses.

Some cases improve and remain free from symptoms for months and even years, but since the majority of reported cases have only been recently observed, time must elapse before it is possible to say with certainty that complete and permanent recovery ever takes place.

On the other hand, the disease is often fatal. Thus of sixty cases collected and analysed by Harry Campbell and myself, twenty-three ended fatally. It is, however, probable for various reasons that these figures exaggerate the fatality of the disease. Although the average duration of life in the fatal cases was about a year and a half from the onset of the disease cases are on record which have lived five, ten, even fifteen years.

In the great majority of fatal cases death has been due to an attack of dyspnœa. The end is usually sudden and often quite unexpected.

Treatment.—Although at the present time no specific treatment is known which will arrest or cure the disease, palliative measures are of great importance.

Since muscular exertion and mental excitement increase the severity of the symptoms, they must both, so far as possible, be avoided. Psychical impressions undoubtedly exert considerable influence on the course of the disease, and I would strongly advise that the patient be given à favourable prognosis. The gravity of the condition should be made known to his friends, and they must clearly understand that the disease is not hysteria. They must neither make light of his symptoms, nor encourage him to fight against the weakness when it appears.

Especial care should be taken to spare the muscles chiefly affected by the myasthenic state. If the symptoms are severe, the patient should be confined to bed. Cold is to be avoided, since it increases the myasthenia.

The feeding of patients suffering from myasthenia gravis requires most careful attention. When there is much weakness of the muscles of mastication and deglutition, the patient should be enjoined to take the larger portion of his food early in the day. All solid food should be minced in order to spare the muscles of mastication, and to avoid, so far as possible, the risk of choking. It is probable that the lives of some of the fatal cases might have been saved had this precaution been attended to, for impaction of an unchewed bolus in the throat has in several cases caused death from asphyxia due to pressure on the windpipe.

Faradism is on no account to be used since it increases the myasthenic state. Massage appears to be of no service. Weak galvanic currents applied to the affected muscles have been said to do good and may be tried.

Although one has seen a number of drugs employed one cannot say with certainty that any had a distinctly beneficial effect on the course of the disease.

According to Strümpell, the attacks of dyspnœa may sometimes be relieved by drawing forward the tongue. In a severe attack in which the patient's life is threatened, artificial respiration should be resorted to. It is, I think, possible that in such a case inhalations of oxygen and transfusion with saline fluid may prove of value.

3. ON A METHOD OF ESTIMATING THE INTERFERENCE WITH THE HEPATIC METABOLISM PRODUCED BY DRUGS

By D. NOËL PATON, M.D., F.R.C.P., Ed., and J. EASON, M.B., C.M., M.R.C.P., Ed.

IT has been for long believed that certain drugs diminish the activity of the chemical changes in the animal body, and the observation that, under the administration of sulphonal, hæmatoporphyrin an imperfectly metabolised katastate of hæmoglobin may appear in the urine, has further directed attention to this matter. But so far, no attempt has been made to determine which tissues or organs are specially affected, or to measure the degree of interference exercised by different agents.

It appeared to us that some indication of the structures acted upon, and of the degree of interference with their functions might be gained by a study of the extent to which the waste nitrogen of the body is elaborated into urea, since it is in the liver that this formation of urea is chiefly carried on and since, therefore, any interference with the activity of this gland should make itself manifest in a diminution in the formation of urea. One of us [1] has already shown that under the influence of diphtheria toxin, the proportion of nitrogen elaborated into urea is diminished, and that during digestion when the hepatic metabolism is more active, the proportion of nitrogen in urea is increased.[2]

With the object of testing this idea, we commenced to study the influence upon the elaboration of nitrogen into urea of some drugs which are supposed to interfere with the normal course of metabolism. Unfortunately, the investigation was interrupted, and we are compelled to make a preliminary communication in the hope that it may induce others to carry out a more extended study of the subject.

Method. — The method adopted was as follows: The perineum of a female dog was split under full anæsthesia to allow of catheterization. The dog was then put on a fixed diet of porridge and milk, or of dog biscuit and milk till it came into nitrogenous equilibrium. It was then subjected to

[1] *Journal of Physiology*, xxiv. p. 331, 1899. [2] *Ibid.*, xxv. p. 443, 1900.

the agent, the influence of which it was desired to study, and again maintained for some days in the normal condition.

The animal was kept in a cage with a zinc floor sloping to a hole in the centre and the urine was collected in a vessel placed beneath or by the catheter. The fæces were formed and were removed as soon after they were passed as was possible. If on any day the urine was contaminated by their presence, it was of course rejected. That this method of collecting the urine is satisfactory if properly carried out, is shown by a comparison of the composition of the urine so collected with urine taken by the catheter.[1]

The quantity, specific gravity and reaction of the urine of the twenty-four hours were determined, and it was diluted to a convenient volume.

The total nitrogen was estimated by Argutinsky's modification of Kjeldahl's method.

The urea nitrogen was estimated by Bohland's method and includes a small amount of ammonia nitrogen.

The non-urea nitrogen was determined by difference.

The ammonia nitrogen was estimated by Schlösing's method.

In some of the experiments the total sulphur and the sulphur in the oxidised form, *i.e.* as sulphuric acid, were determined, the latter by the usual gravimetric method, the former by that method after igniting with caustic potash and nitre.[2]

The phosphates were determined by titration with uranium nitrate.

The observations were for the most part made continuously, but for convenience the record is broken up into separate experiments. The same dog was used throughout the experiments—a setter bitch weighing between 18 and 19 kilos.

Throughout the paper weights are expressed in grms.

I. *Morphine*

Although morphine has so marked an influence on the nervous system, evidence that it affects the general metabolism is wanting. Nor is there any indication that it has a special

[1] *Journal of Physiology*, xxv. p. 445, 1900. [2] *Ibid.*, xxiv. p. 335, 1899.

action upon the liver, for Rutherford found that 3 grains of the acetate had no influence on the secretion of bile.

EXPS. I. and II.—The dog was fed daily on two Spratt's biscuits and 500 cic. of milk. On 25th and 31st July, 0·15 grms. of bimeconate of morphine were given hypodermically at 10 A.M.

	Nitrogen			Sulphur			Phosphorus as		Chlorine as
Date	Total	Urea N	Urea N % of total	Total as SO_3	Oxidised as SO_3	Oxide % of total	P_2O_5	P_2O_5 per 100 N	NaCl
23. vii.	5·06	4·64	91	...	·408	...	...	...	...
24. ,,	8·33	7·86	94	...	·689	...	1·84	22	1·96
25. ,,	9·82	8·86	90	1·14	·652	57	1·90	19	2·04
†26. ,,	4·67	4·28	91	0·61	·346	56	0·72	15	1·20
27. ,,	8·56	7·61	89	1·22	·696	57	1·82	21	2·00
28. ,,	8·41	7·70	91	1·21	·612	56	1·84	22	2·48
31. ,,	6·13	5·76	94	...	...	...	0·78	12	...
1. viii.	8·51	7·74	90	...	...	...	1·74	20	...
2. ,,	6·16	5·32	86	...	...	...	1·22	19	...

† Only one biscuit given.

Average per cent. of nitrogen in urea.

Before	First morphine	After	Second Morphine	After
93	90	90	94	86

The Spratt's biscuits are somewhat irregular in weight, and the dog was not in a condition of steady nitrogen balance, but this in no way interfered with the object of the experiment. Morphine, in these doses, though producing marked nervous symptoms, does not seem to interfere with the hepatic metabolism.

A point of some interest is the fall in the proportion of phosphorus as phosphoric acid to the nitrogen.

II. *Sulphonal*

The appearance of hæmatoporphyrin in large quantities in the urine after the administration of sulphonal has been taken to indicate a profound interference with the chemical changes in the body and probably with the hepatic metabolism which is more especially concerned in the elaboration of the normal urinary pigment.

EXP. III. Same dog. Diet: oatmeal 500 grms., milk 700 c.c., water, 1500 c.c.

Nitrogen of Urine

Date.	Total.	Urea N.	Ammonia N.	Urea N. p.c. of total.	Ammonia N. p.c. of total.
3. xi.	11·76	9·91	...	84	...
4. ,,	11·08	9·07	0·95	82	8·5
5. ,,	11·92	9·91	0·97	83	8·1
6. ,,	12·76	11·42	1·05	89	8·2
7. ,,	12·76	10·42	1·11	82	8·6
*8. ,,	13·10	10·86	0·84	83	6·4
*9. ,,	12·43	9·08	...	73	...
10. ,,	11·70	8·77	0·90	75	7·6
11. ,,	...	...	...	...	...
12. ,,	9·84	7·62	0·78	77	7·9
13. ,,	12·82	10·25	1·18	80	9·2
14. ,,	13·83	10·92	...	79	...

* 3 grms. sulphonal on each day.

Average per cent. of nitrogen in urea.

Before.	Sulphonal period.	After.
84	74	79

Decrease 9 per cent.

This experiment seems to show that sulphonal markedly interferes with the elaboration of nitrogen into urea.

We have examined the proportion of urea nitrogen in a specimen of urine from a case of sulphonal poisoning sent to us by Dr Easterbrook. The following figures were obtained:

Total Nitrogen 1·91 per cent.

Urea Nitrogen 1·65 per cent. = 86 per cent. of total.

In the five experiments recorded by Dunlop, Macadam, and Noël Paton,[1] the urea nitrogen constituted from 91 to 95 per cent. of the whole, and if these figures are accepted as an average there was thus a diminution in the nitrogen in urea in this case. But it must be remembered that 86 per cent. is not below the average as determined by Gumlich[2] and Bohland.[3]

III. *Coal Gas*

The general diminution in the processes of oxidation in the body produced by carbon monoxide is well known: but how far this affects the hepatic metabolism has not been investigated.

[1] *Journal of Physiology*, xxii. p. 69, 1897.
[2] *Zeitsch. f. phys. Chem.*, xvii. p. 10, 1892.
[3] *Pflüger's Arch.*, xliii. p. 30, 1888.

Araki[1] showed that the administration of carbon monoxide causes the appearance of sugar in the urine.

Exps. IV. and V. Same dog. Same diet as in Exp. III.

	Nitrogen in Urine.								Phosphorus.		Sulphur.		
	Total.		Urea N.		Ammonia N.		Urea N. p.c. of total.	Ammonia N. p.c. of total.					
Date.	Per cent.	Per diem.	Per cent.	Per diem.	Per cent.	Per diem.			as P_2O_5.	P_2O_5 in 100 N.	Total as SO_3 per diem.	Oxidised as SO_3 per diem.	Oxodised S p.c. of total.
Exp. IV.													
13. xi.	·641	12·8	·512	10·2	·059	1·18	80	9·2	...	...	...	...	...
14. ,,	·692	13·8	·546	10·9	·045	0·91	79	6·5	...	...	...	...	...
15. ,,	...	...	...	...	...	...	...	...	...	...	...	...	...
16. ,,	·526	...	·414	...	...	...	78	...	...	...	...	...	...
*17. ,,	·456	...	·341	...	·035	...	75	7·6	...	...	...	...	...
Exp. V.													
19. xi.	·428	12·8	·358	10·7	·028	0·84	83	6·5	...	...	...	...	...
20. ,,	·415	8·3	·341	6·8	·037	0·74	80	8·9	...	...	1·64	1·08	66
*21. ,,	·574	11·5	·462	9·2	·033	0·66	80	5·8	2·96	28	2·46	1·72	69
22. ,,	·585	11·7	·442	8·8	·049	1·00	75	8·6	3·40	29	2·50	1·36	54
23. ,,	·644	12·9	·498	9·9	·048	0·96	77	7·5	3·40	27	2·78	1·56	56
24. ,,	·644	12·9	·521	10·4	·043	0·86	80	6·7	3·34	26	2·70	1·68	62
25. ,,	·622	12·4	...	...	...	...	...	...	...	...	...	...	...
26. ,,	·521	10·4	·414	8·3	...	...	80	...	3·20	30	...	...	...
27. ,,	·607	12·0	·476	9·5	·039	0·69	79	6·3	3·26	27	2·10	1·56	74

The urine was lost on the 15th and was not analysed on the 18th. On the 16th and 17th some urine was lost and the percentage analyses alone are given.

* On the 17th the dog was placed in a closed chamber with coal gas till it became drowsy, and on the 21st in the same chamber till it was deeply narcotised.

Average per cent. of nitrogen in urea.

Before.	During.	After.
79	75	81
81	75	79

Diminution of 6 per cent.

[1] *Zeitsch. f. phys. Chem.*, xix. p. 459, 1894.

These observations show that the coal gas diminishes the proportion of nitrogen in urea and of sulphur in the oxidised form, but that it does not alter the proportion of phosphorus to nitrogen.

IV. *Alcohol*

It has been generally recognised that alcohol in large doses has a markedly toxic action on the metabolism but its influence on the hepatic function has not been specially studied. That it has a special action on the liver, seems to be indicated by the part it plays in the etiology of cirrhosis of the organ. Reiss[1] states that it diminishes urea and increases uric acid, while Schmiedeberg and Hofman[2] found that it hindered the synthesis of hippuric acid.

EXP. VI. Same dog. Same diet as in Exps. IV. and V.

Nitrogen of urine

Date	Total N	Urea N	Urea N p.c. of total	
24. xi.	12·88	10·42	80	
25. ,,	12·43	...	...	
26. ,,	10·42	8·29	80	
27. ,,	12·04	9·52	79	
28. ,,	12·66	9·52	79	
29. ,,	10·02	7·28	73	60 c.c. absolute alcohol at 10.
†30. ,,	8·12	5·94	73	150 c.c. ,, ,, ,,
1. xii.	Fæces in urine		...	
2. ,,	,, ,,			
3. ,,	,, ,,			
4. ,,	10·08	7·95	79	
5. ,,	11·54	9·52	82	
6. ,,	11·59	9·07	78	

† Dog appeared intoxicated.

Average per cent. of nitrogen in urea.

Before	Alcohol	After
79·5	73	79·6

Diminution of 7·5 per cent.

V. *Quinine*

Upon the influence of quinine on the metabolism a very large amount of work has been accomplished which is admir-

[1] *Zeitsch. f. klin. Med.*, 1880.
[2] *Arch. f. exp. Path. u. Pharm.*, vii. p. 243, 1877.

ably summarized in Wood's *Therapeutics*, 7th edition, 1898, p. 578. There seems little doubt that it does diminish the disintegration of proteids and there is considerable evidence that it has a special action in inhibiting the production of uric acid. But so far its action on the general distribution of nitrogen in the urine has not been studied.

Zuntz[1] was unable to find any influence on the absorption of oxygen or the elimination of carbon dioxide.

We have performed but one experiment and in this only ·75 grms. of quinine were administered. The result was negative.

CONCLUSIONS

The observations given above are too few to allow certain conclusions to be drawn, but those which they suggest are as follows:

1. Certain drugs have a marked effect on the elaboration of the waste nitrogen into urea while others do not act in this way, (*a*) sulphonal, alcohol and coal gas (carbon monoxide?) have this action, (*b*) quinine in small doses and morphine in large doses do not manifest it.

2. Under the influence of coal gas the percentage of oxidised sulphur in the total sulphur of the urine is diminished concomitantly with the diminution in the percentage of nitrogen in urea, while under morphine the percentage of oxidised sulphur as well as the percentage of urea nitrogen are unchanged, thus bearing out the conclusions as to the relationship of these processes arrived at by one of us.[2]

3. It is probable that the diminution in the proportion of nitrogen in urea and of sulphur in the oxidised condition affords an indication of the interference with the hepatic metabolism produced by any drug.

4. While the administration of morphine causes a diminution in the proportion of phosphorus to nitrogen in the urine, coal gas (carbon monoxide) has not this action.

[1] *Arch. f. Phys.*, p. 203, 1894. [2] *Journal of Physiology*, xxiv. p. 335, 1899.

Meeting VII.—May 1st, 1901

MR A. GORDON MILLER, *President, in the Chair*

The following reply to the Address to His Majesty the King was received from Lord Balfour of Burleigh :—

SCOTTISH OFFICE,
WHITEHALL, 30*th March* 1901.

SIR,—I am commanded by the King to convey to the Medico-Chirurgical Society of Edinburgh His Majesty's thanks for the expressions of sympathy with the Royal Family on the occasion of the lamented death of Her late Majesty, Queen Victoria, and for the loyal and dutiful assurances on the occasion of His Majesty's accession to the throne contained in their Address, which I have had the honour to lay before His Majesty.—I am, Sir,

Your obedient Servant,
BALFOUR OF BURLEIGH.

THE PRESIDENT,
EDINBURGH MEDICO-CHIRURGICAL SOCIETY,
EDINBURGH.

I. ELECTION OF MEMBERS

G. R. Livingston, M.B., C.M., Dumfries ; A. Dingwall Fordyce, M.B., Ch.B., 34 Gt. Stuart Street ; J. A. Thomson, M.B. Ch.B., Craiglockhart Hospital ; and George Dickson, M.D., 14 Ardmillan Terrace, were elected Ordinary Members of the Society.

II. EXHIBITION OF PATIENTS

Dr Norman Walker exhibited—

(*a*) A case of BAZIN'S DISEASE. The girl, aged 16, came to the Infirmary about eight weeks ago with an eruption on one leg, extending up to the waist, which looked something like Ecthyma. The lesions were, however, deeper, and had a more chronic appearance than the lesions of that disease, and all cultivations from them proved to be sterile. The girl has markedly enlarged cervical glands and some dulness at one apex, with cough and expectoration. The

case at that stage was the first definite example he had seen of the condition described by Kaposi as Acne Scrophulosorum. During her stay in hospital there was no great improvement, but soon after she went home the condition got much worse, and she returned with the lesions advanced, and the condition now closely resembles that known as Bazin's Disease.

(*b*) Two other cases which he showed mainly for the interest attached to their treatment. The first was a case of Lupus, of very extensive distribution, which he had treated for the past five months with weekly applications of the X-Rays, latterly the exposure lasting for twenty minutes. The patient was far from well, but the improvement was very marked, and the increase in comfort was alone worth the trouble.

During a recent visit to the London Hospital, where one has an opportunity of seeing both varieties of the Light treatment, he was greatly impressed by the success attendant on the much less advertised X-Rays treatment. While there on a previous occasion he had seen a further use of the Rays in connection with the treatment of Rodent Ulcer, the results of which were so good that it was almost necessary to see to believe. Large cavities, occupied by inoperable cancers, had filled up and healed with a smooth scar. The treatment had been commenced on two of Dr Jamieson's patients, and he showed one of them who had had six exposures to the Rays.

III. Exhibition of Instrument

M. Shaw M'Laren exhibited and demonstrated the use of Schlagintweit's Catheter-Cystoscope. It consists of two parts: first, an ordinary metal catheter, carrying an electric lamp at its point; and second, the telescope part of the ordinary cystoscope, which is pushed down into the catheter so that the prism lies in the eye of the catheter, and exactly fits and closes it. The catheter part is introduced into the bladder in the usual way; the bladder can be washed out and filled with clean fluid, and then, *without further change*, the telescope tube is pushed in, and the examination proceeds. The bladder can be re-cleaned as often as required through the instrument, which remains in position. The advantages are enormous, as the bladder suffers the minimum of irritation, and there is no risk of setting up

fresh bleeding as so often happens with the ordinary cystoscope. It is also fitted with an admirable arrangement by which it can be connected with a "douche-can," and with a small siphon collector below for showing when the fluid is clear.

IV. Demonstration

Dr Paterson exhibited a series of naked-eye specimens, mounted in glycerine jelly, illustrating the pathology of Eye Diseases; also a large number of lantern slides dealing with the same subject. The results of various traumatic and inflammatory lesions were illustrated and commented on. In speaking of optic neuritis, Dr Paterson declared himself a supporter of the old view that the typical choked disc of cerebral tumour is an œdema, and not a true neuritis. Specimens were shown in support of this view, in which the distension of the optic nerve sheath and the thickening of the arachnoid were enormously developed. A large number of intra-ocular tumours were shown in various stages of growth. One of these, an angio sarcoma, of the iris showed some very peculiar histological characters, very few similar growths having been described.

V. Original Communication

ARTERIAL HYPERTONUS & ARTERIO-SCLEROSIS: THEIR RELATIONS AND SIGNIFICANCE.

By William Russell, M.D., F.R.C.P. Edin., Assistant Physician, Royal Infirmary, Edinburgh.

Introductory.—I propose to ask your attention to-night, first to the various applications of the term "arterio-sclerosis"; secondly, to my own observations on the condition; thirdly, to a state of the arteries to which I have ventured to apply the term "hypertonus"; and fourthly, to the relations of hypertonus to arterio-sclerosis. "Arterio-sclerosis" is not a term of Anglo-Saxon birth; it has come to us from our brethren on the continent of Europe. It is only within recent years that it has come into general use in this country, and if proof of this were

wanted it is to be found in the fact that the word does not even occur in the first edition of Fagge's "Principles and Practice of Medicine," published in 1886, or in the sixth edition of Bristowe's "Treatise on the Theory and Practice of Medicine," published in 1887. If those who use the term are asked to define it a considerable variety of reply is given, and it becomes evident that a varied significance attaches to it. Some mean atheroma, others mean fibrous thickening, others mean degeneration, and others mean a combination of all three conditions. If there is any one characteristic which is recognised by all those who use the term, that characteristic is thickening or induration of the arterial walls as tested by the condition of the radial artery. Such vague and indefinite notions regarding a common condition are at least unsatisfactory, and as I have had considerable opportunities of studying the condition a brief account of my observations may be of some help in giving the condition a more definite position. Before recording these observations it is, however, necessary to deal with what we may regard as the authoritative definitions and explanations of the term in question.

Historical and Pathological.—A historical *résumé* entitled "Étude Clinique sur les Indurations des Artères," by Dr Nöel Gueneau de Mussy, appeared in the *Archives Générales de Médecine* of 1872, p. 129. Dr de Mussy showed that the condition is common in early and middle life and not necessarily present in old age; and he included atheroma under the term "induration." Ziegler says that "sclerosis in an artery implies the existence of local thickening of its inner coat," and proceeds then to describe atheroma. He was the first to describe in detail and to discuss arterio-sclerotic atrophy of the kidney. He showed that the renal arteries and their branches in aged people are very frequently the seat of sclerotic changes which may simultaneously affect the arteries of other regions also or be confined to the kidney. In this condition the intima of the arteries is notably thickened and the lumen correspondingly narrowed or even obliterated; and he showed that this vascular change in the kidney gave rise to a special form of contraction of the organ which he termed "arterio-sclerotic atrophy."

All the German pathologists may be said to use the term "arterio-sclerosis" to describe atheroma, whatever else they may

include under it. Of all the German work, however, there can be no question that it is Thoma's which has specially influenced opinion in this country and in America. To borrow a term from social life, he may be said to have "set the fashion" in the use and interpretation of the term in clinical medicine and, so far as we have been able to find, pathology has not let its voice be heard beyond the lecture-room. Thoma's views may be briefly summarised as follows: the elasticity of the vessel wall becomes reduced in general diseases, acute and chronic infective diseases, long-continued disturbances of general nutrition, by many poisonous substances, and by functional overstraining of arteries from an increase of the heart's action. As a result the lumen dilates and the blood stream is retarded in the widened vessel. This retardation is the cause of a new formation of connective tissue in the intima of the widened artery. "The new formation of connective tissue in the intima renders the vessel wall more firm so that it appears more rigid and less yielding (arterio-sclerosis, phlebo-sclerosis, angio-sclerosis)."[1] In short, Thoma holds that the thickening of the intima is secondary to a dilatation of the vessel and consequent on slowing of the blood-stream; that the thickening is to compensate for the assumed widening and to restore the equilibrium between lumen and contained blood which had been lost. By this means, he maintains, the rapidity of the blood flow is re-established.

If we turn to the French school we find Lancereaux in 1893 writing on "L'Endartérite ou Artério-sclérose Généralisée."[2] He defines the conditions as a proliferation of the cells of the intima which goes on to fatty degeneration due to failure of nutrition. Huchard[3] mentions the fact that German authors always use the term arterio-sclerosis to signify atheroma of arteries, and he asks whether the terms are absolutely synonymous. His answer is strongly in the negative. He uses the term to designate a general condition of which atheroma is only one of numerous manifestations. These include visceral scleroses—all dominated by a primary chronic inflammation of the small vessels described by many authors as obliterative endarteritis (or endarteriolitis). Arteritis of the

[1] Pathology and Pathological Anatomy, English Translation, p. 247.
[2] Archives Générales de Médecine, 1893, I., pp. 5 and 164.
[3] Maladies du Cœur et des Vaisseaux, 2ème édition, Paris, 1893.

small vessels, in short, he considers the anatomical characteristic of arterio-sclerosis (p. 96).

Turning to English works of recent date it is not necessary to go beyond the valuable "System of Medicine" which has been edited by Dr T. Clifford Allbutt. Here we look for the high-water mark of contemporary medicine, and in Dr F. W. Mott we find an exponent of the German view of arterio-sclerosis and an adherent of Thoma's views which have already been referred to. He places the term "arterio-sclerosis" as the title of the section and in the introduction to the subject he says that the term "is applied rather loosely to a thickening of the vessel-wall. It includes the obvious naked-eye change in the large arteries, named by some authors atheroma; by others, endarteritis deformans. It includes also arterio-capillary fibrosis, a change first described by Gull and Sutton in the walls of the small vessels, which only becomes obvious on microscopic examination." At page 320 he gives as his definition of arterio-sclerosis "a local or general thickening of the arterial wall with loss of elasticity, occasioned mainly by fibrous overgrowth of the tunica intima, secondary and proportional to weakening of the muscular and elastic elements of the media." It seems quite clear that Dr Mott includes atheroma under the term arterio-sclerosis, and he follows Thoma in asserting that the primary change in atheroma is to be found in a degeneration of the tunica media and that the thickening of the tunica intima is secondary and compensatory to this. He holds with Thoma that the observations made by the latter on paraffin moulds of the aorta prove this without apparently seeing that these moulds may, with equal justice, be held to prove that the atrophy of the media is secondary and compensatory to the hypertrophy of the intima. It is not my purpose at present to elaborate this point, but I would indeed be surprised to find that British pathologists held this to be the order of events in atheroma. For myself I may say that I have no doubt whatever that atheroma is a primary affection of the tunica intima, degeneration and destruction of the media being secondary; in fact, atheroma is so entirely a process by itself that it ought to be allowed to stand in its old position; it seems to me to be quite unnecessary to extend to it the German use of the word "arterio-sclerosis."

Passing from this question of the inclusion of atheroma in

arterio-sclerosis we find that Dr Mott describes a "diffuse arterio-sclerosis" (p. 329) in which the changes begin in the small arteries and capillaries, especially those of the renal cortex, brain, and heart, and are frequently associated with "nodular atheroma" of the aorta. He says that "on minute examination the muscle fibres of the media show hyaline swelling, fatty degeneration, or atrophic changes so that the muscular elements are often not recognisable; this is especially the case in the small arteries of the kidney where the wall of the vessel may appear to consist of a homogeneous hyaline tissue. Sometimes the degenerated atrophied fibres of the media can be made out, but nothing of the elastic lamina, the intima being thickened and represented only by a homogeneous hyaline material with but few nuclei." The result of these widespread changes is increased resistance to flow of blood through the capillaries, hypertrophy of the left ventricle, dilatation of larger arteries from degenerative changes in muscular and elastic tissues of the media, slowing of the circulation, and compensatory proliferation of the subendothelial layer of the inner coat" (p. 330). Here, again, Dr Mott strongly commits himself to Thoma's view. In America Osler, Councilman, and others have committed themselves to Thoma's views.

From the foregoing *résumé* which while not professing to be exhaustive, may be taken as fairly representative of authoritative views, it is seen that the term arterio-sclerosis is applied to three conditions: (1) to atheroma; (2) to a generalised endarteritis; and (3) to a thickening of the intima compensatory to dilatation of vessels from weakening of their middle coat. This *résumé* could not, however, be regarded as even approximately complete without some reference to the views, expounded by men of eminence, on the relations between granular kidney and the changes in the arteries and heart which accompany granular kidney. In fact, no exposition on arterial changes could suitably ignore this side of the question.

George Johnson (1850-1873) was the first to show that thickening of the muscle coat of the arteries occurred in granular kidney, and he held that the changes in the heart and vessels were secondary to primary kidney changes. Gull and Sutton (1872-1877) propounded the view that there occurred a generalised change or disease in the vessels, to which they gave the name "arterio-capillary fibrosis." The

changes in the kidney were regarded as secondary to this vessel fibrosis. Dickinson (1875) held that a growth of fibrous tissue in the kidney was the primary change and that the changes in the vessels were secondary to this. Rosenstein (1881) agreed with Johnson as to the thickening of the muscular coat of the arteries, but thought that the thickening of the intima was inflammatory. Ewald (1881) held that there were two forms of changes in the vessels connected with Bright's disease: first, that in which the kidney was affected primarily and was followed by hypertrophy of the heart and of the muscle coat of the arteries; and, second, that in which disease of the general vascular system was the starting-point of the kidney disease. In this latter there was arterio-capillary fibrosis both in the kidney vessels and in the general vascular system. He thought that it was a question whether such cases should be called "Bright's disease" or not. Dreschfeld and Mahomed (1881) held practically the same view as Ewald, the latter holding in addition that there might be cardio-vascular changes without necessarily renal changes being present in all. Samuel West carried the discussion down to last year and discourses on the old theme as to whether the kidney lesion or the arterial change is the first to appear. He enters a protest against granular kidney being considered partly under vessels and partly under kidney.

Personal Observations.—I now ask your attention to my own observations which were conducted on a simple plan. All the cases I examined were seen by me during life. As the radial artery is the vessel which is taken as the type during life I decided to examine it after death. I also usually examined the renal artery, the coronary artery, and the kidney itself. In one case the cerebral nutrient vessels were examined by my friend Dr Ford Robertson. In this way I have examined so far sixteen cases and the results I have arrived at may be stated as follows: 1. Atheroma and arterio-sclerosis are two totally distinct clinical and pathological entities; 2. Atheroma is a localised and patchy affection of the arteries, characterised by degenerative changes which have long been recognised. 3. Arterio-sclerosis is a generalised affection of the arteries and is characterised by (*a*) thickening of the tunica media, this thickening being primarily a true hypertrophy, although it may ultimately show some degeneration; (*b*)

thickening of the tunica intima from fibrous hyperplasia of the subendothelial connective tissue without atheromatous degeneration; and (*c*) in some instances fibrous thickening of the tunica adventitia. 4. The changes in the arteries *in* the kidneys differ from those in the radial, or even in the renal arteries themselves before they enter the kidneys. They differ in the following respects: in the kidney the thickening of the intima is proportionally greater, the media is not appreciably thickened, it may even be atrophied and may have undergone hyaline degeneration. (The atrophic changes in the kidneys are in proportion to the sclerotic changes in the vessels.) 5. The lumen of the radial arteries and of the arteries in the kidneys is markedly diminished. 6. The changes in the nutrient arteries of the brain, and probably of the cord, correspond with those in the arteries inside the kidneys. 7. Arterio-sclerosis may be associated with more or less atheroma in the same subject.

My cases contradict Thoma's view that the thickening of the intima is compensatory to weakening and yielding of the media. In only one of my cases would the condition of the radial artery lend any support to this view. Fig. 1 represents the radial artery in this case, and it will be seen that the vessel has a large lumen, that the muscle coat shows degeneration, and that the intima is thickened. Although this was the condition of the radial artery the changes in the kidney vessels corresponded with the changes in my other cases. Fig. 3 and Fig. 5, which are typical examples, represent the radials from two of my cases, and show the small lumen, the thickened media, and the thickened intima. Fig. 4 represents the kidney from the same case as Fig. 3, and shows the marked thickening of the intima without thickening (perhaps even some atrophy) of the media. Fig. 6 represents the kidney from the same case as Fig. 5, and shows the great thickening of the intima, going on to occlusion of the lumen of the vessel, and along with this the atrophy and disappearance of the media as a distinct structure. This case is the one in which Dr Ford Robertson examined the nutrient arteries in the brain, and the changes in them correspond with the changes here described in the kidney.

In only one of the series which I have examined was there atheroma of the radial artery, and in none was there atheroma

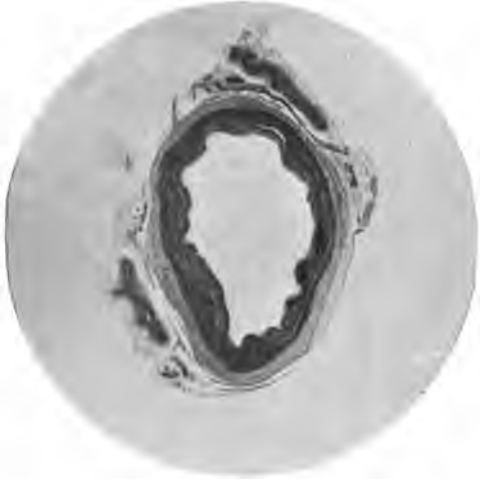


FIG. 1.—Radial artery showing large lumen, degeneration of media, and thickening of intima. × 16 diameters.

FIG. 2.—Radial artery showing hypermyotrophy. × 16 diameters.

TO ILLUSTRATE DR W. RUSSELL'S PAPER.

FIG. 3.—Radial artery showing small lumen, thickened media and intima. × 16 diameters.

FIG. 4.—Kidney from same case as Fig. 3, showing sections of two arteries with much thickened intima without corresponding thickening of media. × 50 diameters.

TO ILLUSTRATE DR W. RUSSELL'S PAPER.

FIG. 5.—Radial artery showing small lumen and thickened media and intima. × 16 diameters.

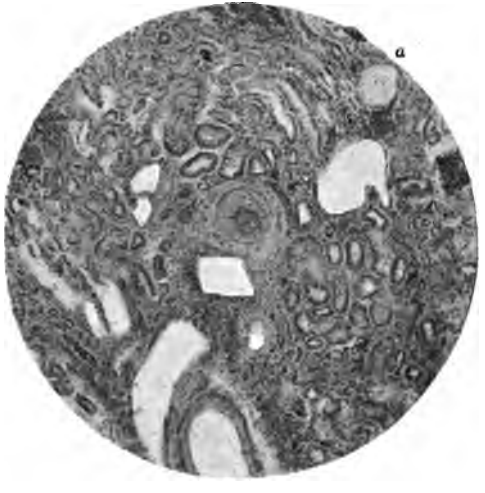


FIG. 6.—Kidney from same case as Fig. 5, showing in centre of field artery with greatly thickened intima and atrophied media. At *a* occluded arteriole. × 50 diameters.

TO ILLUSTRATE DR W. RUSSELL'S PAPER.

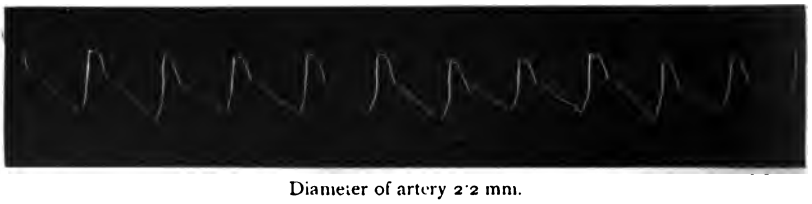
TRACING 1 (CASE 1).

Diameter of artery 2·3 mm.

TRACING 2 (CASE 1).

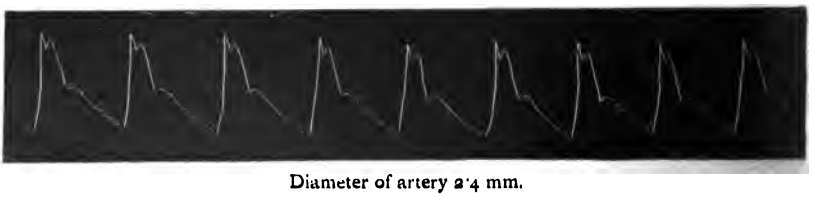
Diameter of artery 2·7 mm.

TRACING 3 (CASE 2).



Diameter of artery 2·2 mm.

TRACING 4 (CASE 2).



Diameter of artery 2·4 mm.

TO ILLUSTRATE DR W. RUSSELL'S PAPER.

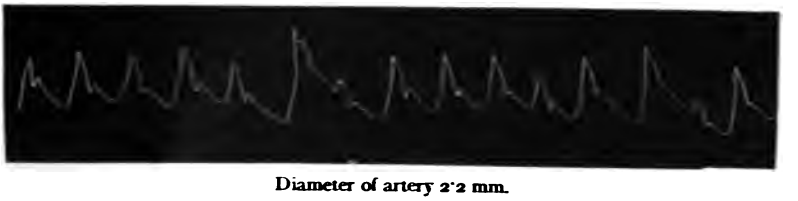
TRACING 5 (CASE 3).

Diameter of artery 2·4 mm.

TRACING 6 (CASE 3).

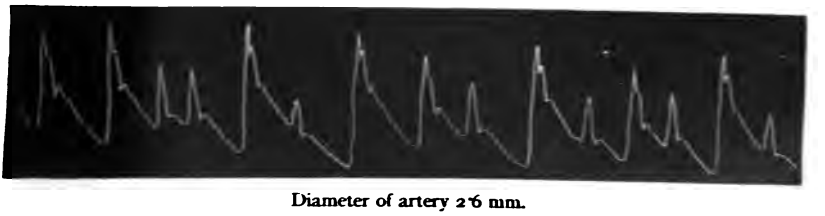
Diameter of artery 2·7 mm.

TRACING 7 (CASE 4).



Diameter of artery 2·2 mm.

TRACING 8 (CASE 4).



Diameter of artery 2·6 mm.

TO ILLUSTRATE DR W. RUSSELL'S PAPER.

of the kidney vessels. In some of the cases there was hardly any atheroma of the large arteries. The thickening of the intima in the arteries of the kidney goes on to complete occlusion of the vessels affected; it cannot, therefore, be compensatory in the sense in which Thoma and his followers apply the term. Were it compensatory it should cease with the establishment of compensation.

Much of the confusion and difference of opinion which characterise writings on the kidneys and arteries have, I think, arisen from a failure to separate the points of similarity and of dissimilarity in the changes which occur in the arteries inside and outside the kidney. Inside the kidney the changes are most marked in the intima; outside it the changes are most marked in the media. In both, however, the intima is thickened. Inside the kidney the media may disappear, and it often atrophies; outside the kidney this never occurs. In two of my cases the change in the radials was almost a pure hypermyotrophy such as Savill described, and it is interesting to note that in one of these there was a diffuse kidney cirrhosis with only slight thickening of the intima of the kidney arteries. Did time permit I might enlarge upon the relations of kidney and arterial change, but this must meanwhile be left. Fig. 2 represents this almost pure hypermyotrophy, and so far as I can at present see this is the only condition which may not be clinically distinguishable, or at least not readily distinguishable from arterio-sclerosis.

Clinical Aspect of Arterio-Sclerosis.—If we now turn to the clinical side of arterio-sclerosis the diagnosis rests primarily upon the condition of the radial arteries, supplemented by observations on the temporal and other vessels of corresponding size readily accessible to observation. The condition of the radial artery is taken as an index of the condition of vessels beyond direct examination. When brain, heart, or renal symptoms are present along with changes in the radial they are referred, in the case of the brain and heart, to the condition of the nutrient arteries of these organs. The changes are presumed to be of the same nature. In a case with renal symptoms the condition of the radial artery forms a large part of the foundation warranting a diagnosis of marked kidney change. The vessel is indurated and its wall is thickened; the induration is uniform, and the vessel, as felt

upwards along its course, has been compared to whip-cord or to other substances. The truest comparison is to be found in the difference which, to any educated finger, exists between a thin-walled and a thick-walled piece of rubber tubing of the size of an average radial artery. The uniformity of this thickening distinguishes the condition from atheroma where the thickening is irregular and may present areas of calcification. This thickened condition of the radial wall is often spoken of clinically as "high tension." This error has arisen from the fact that in high tension the artery feels cord-like, and thus all cord-like pulses are often loosely spoken of as high-tension pulses. The error is a very grave one, for the thick-walled vessel may have a very low pressure blood-stream; and the word "tension" in this connection is used as if it were synonymous with pressure, and as if the thick vessel always, and necessarily, meant high pressure. Indeed, it would be wise to abandon this word "tension" altogether until it came to be properly applied to a condition of the vessel wall alone, and had clearly separated from it any application to blood-pressure. In anything I may say here I shall use the term "blood-pressure" in its correct sense.

Another common clinical error is to speak of a thickened radial artery as atheromatous, there being a widespread idea that thickening in arteries is synonymous with atheroma.

Arterial Tonicity and Hypertonus.—In the next place I wish to emphasise a feature of the pulse—including therein, as is commonly done, the condition of the arterial wall as well as the blood wave—which is largely ignored. I refer to the state of contraction of the vessel wall. Relaxation is recognised, in acute pneumonia for instance, and its significance is not overlooked, but is regarded as an indication for certain therapeutic measures. Arterial spasm, on the other hand, is recognised in angina pectoris and in some renal and other diseases. With the exception, however, of certain definite maladies, of which those mentioned are examples, there is little or no attention directed to this factor. The fact of a varying degree of contraction in arteries is really not open to question, and the point does not require to be elaborated; in fact, I think that it is the recognition of this which has led to the confusion of the terms "tension"

and "blood-pressure." In physiological conditions of perfect health when the arteries are tightened up, that is, contracted beyond their ordinary, the blood-pressure is raised because the vigour of the left ventricle is unimpaired. This heightening of blood pressure inside a vessel necessarily, then, implies an increase of tension of its wall. On the other hand, a vessel may be tightened up without the blood-pressure being raised. This occurs when there is an accompanying diminution in ventricular force. In a certain sense even this tightening up of a vessel may be spoken of as "tension" (quite apart from the pressure exerted on its wall by heightening of blood-pressure), but the word is not used in medicine in this sense, otherwise extra contraction of all involuntary muscle, by nervous or other influences, would be spoken of as "tension," whereas "spasm" is the word used.

Spasm, it must be remembered, is only an exaggeration of the normal condition in muscle which is known as "tone" or "tonicity," and it is to this exaggerated tonicity in the vessels that I have ventured to apply the term "hypertonus." The word "spasm" has in medicine too restricted an application to make it suitable for my present purpose. The condition which I have in view is a prolonged or oft-recurring increase of the normal degree of contraction which vessels have in virtue of their muscle coat, and which is physiologically called "tonus." Hypertonus carries with it an important influence on the circulation apart from any alteration in blood-pressure—namely, a diminution in the blood-containing capacity of the vessels from a diminution in their internal diameter. This diminution in lumen necessarily occurs when an active contraction of the vessel wall is present. In diseased conditions it has very important results and determines symptoms of grave significance, to which reference will presently be made.

Recognition of Hypertonus.—We have next to inquire how this condition of hypertonus can be recognised clinically. In the first place it can be either recognised, or suspected, by the educated finger. I have recognised the condition for years and acted on its recognition. With the assistance of my friend Dr Oliphant Nicholson I have, however, been able to demonstrate the existence of this condition. I took cases where by means of the finger alone I judged that

there was this recurring hypertonus. I argued that, if I were correct, the radial artery ought to give a diameter varying with the degree of tonus, and this is precisely what we found to be the case. The measurements were made by means of Oliver's arteriometer. By this instrument the diameter of an artery can be determined, and we found that when the vessel to my sense of touch was in the condition of hypertonus its lumen was smaller than when my finger told me that the hypertonus had passed off. We further took sphygmographic tracings from the artery and found that the arteriometer measurement and pulse-tracing helped to explain each other. I do not, however, wish it to be thought that arteriometer and sphygmograph are necessary to enable the clinician to recognise this condition, for they are not.

To illustrate the existence of hypertonus I direct your attention to sphygmographic tracings and arteriometer measurements from four cases.

CASE I.—A man, aged 35 years, came to me at the Edinburgh Royal Infirmary complaining of headache and general weakness and insomnia. He was a strongly built, well-nourished man, but was somewhat pale. He had been working at his occupation for three months and before that time had been well. The radial artery was markedly thick, the thickening being uniform and extending right up the arm. In fact, it presented the characters which would commonly be described as "arterio-sclerotic." The man was young, and this, combined with his symptoms and the absence of any evidence in the urine of renal disease, led me to surmise that this vessel condition was largely one of hypertonus and that, even assuming that there might be some degree of thickening, his complaints were due to the hypertonus and to the causes producing it. An arteriometer measurement and a sphygmographic tracing were taken and he was admitted into Ward 23. Some doses of erythro-tetranitrate demonstrated that hypertonus was accountable for most, but not all, of the radial thickening; this substance, as is well known, relaxes arterial spasm, but does not, of course, remove an organised thickening, and in this case it greatly reduced the thickening by relaxing the spasm. The arteriometer measurement and the sphygmographic tracings can be compared with the former ones. Tracings 1 and 2 were taken before and after giving erythro-

tetranitrate and the arteriometer measurements were 2·3 and 2·7 millimetres. The existence of hypertonus in this case is of special interest, as we found afterwards that the man had had syphilis.

CASE II.—The next tracings were also from a patient in Ward 23 suffering from spasmodic asthma. He was a man, aged 53 years, and it was quite clear that when the asthmatic spasm supervened his radial arteries became markedly tightened up. The following are the pulse tracings (Tracings 3 and 4) during the asthmatic spasm, and after its relief by means of liquor trinitrina the arteriometer measurements were respectively 2·2 and 2·4 millimetres.

CASE III.—The next tracings (Tracings 5 and 6) were from a man, aged about 60 years, also in Ward 23, suffering from extreme orthopnœa, believed to be of cardiac origin and due to myocardial degeneration. The vessels were thickened, but they also seemed to me to be in a condition of hypertonus. He got great relief from the administration of anti-spasmodics. The arteriometer measurements were respectively 2·4 and 2·7 millimetres.

CASE IV.—The next tracings are from an old man, aged 70 years, who was under my observation for years in Queensberry House. He was subject in his later years to recurring attacks of "renal asthma," during which his permanently-thickened vessels quite appreciably tightened up and became thicker. The sphygmograph gave the following traces (Tracings 7 and 8) during the attacks and in the intervals, and the arteriometer gave the diameter of the vessel as 2·2 and 2·6 respectively.

In these four cases the difference in the pulse when tightened and not tightened was readily appreciable by the finger. The sphygmograms bear out this, while the arteriometer reading of the diameter of the radials under the two conditions explains the sphygmograms. Under hypertonus the swing of the lever is less, the percussion stroke is shorter and less abrupt, the summit tends to be more rounded, and the predicrotic notch less evident. When the hypertonus passes off all this is altered.

Cases in which hypertonus occurs.—Hypertonus occurs in normal arteries and in sclerosed arteries. It occurs in the young, the middle-aged, and the old. In fact, I would specially

impress upon you the fact that even in thickened arteries, showing arterio-sclerosis, this tightening up which I have called "hypertonus" occurs. This combination was present in three or four of the cases from which the pulse tracings were taken. Now if this not only can, but does, occur, the idea of muscular degeneration, with which the literature or arterio-sclerosis is saturated, and consequent inability on the part of the artery materially to respond to nerve or blood influences must be materially modified. Not only this but the recognition of this element or factor of hypertonus in sclerosed arteries is, indeed, of supreme clinical importance. Did time permit I could illustrate this point with numerous cases with symptoms referable in one case to the brain, in others to the spinal cord, heart, lungs, or kidney. In all the symptoms are mainly attributable to the hypertonus lowering the blood-supply to organs which are already badly supplied by the sclerosed arteries. I shall only briefly refer to three cases.

CASE V.—The patient was aged 70 years. He had markedly thickened arteries, arcus senilis, and no albumin. He began to be unusually somnolent during the day, his gait became somewhat unsteady and his speech somewhat thick,' he could not concentrate his mind, and he noticed that in writing letters he misspelt common words. His arteries were tightened up but the blood-pressure was not high. Under treatment directed to the relief of hypertonus the symptoms entirely passed off and he has continued in good health for nearly two years.

CASE VI.—The patient was a very old but active man who had been rather out of sorts for a week or two. He had thickened arteries and no albumin. He suddenly one day became delirious and was kept in bed. His arteries were tightened up without any increase in his blood-pressure. Under treatment directed to the relief of the hypertonus the delirium became daily less and finally disappeared.

CASE VII.—The patient was a man, aged 60 years, who had been under my observation in Queensberry House since July 1899. When admitted he had very thick vessels, but there was no albumin in the urine. After admission he had several attacks of "uræmic coma," in each of which I thought he would die. Although his vessels were amongst the thickest I had seen, arteriometer measurements confirmed my observa-

tion that even they varied in diameter. Acting on the same lines this man recovered and has not had an attack for over a year.

Cause of hypertonus.—We now come to the question of the cause of hypertonus. The condition can, and does, occur at all ages. In strong people it is associated with, and part of, heightened blood-pressure and a pulse of true high tension. In aged people with a relatively feeble or failing heart hypertonus does not lead to an increase in blood-pressure but to the reverse. In fact, it is not infrequently the precursor of heart failure. At all ages the condition, in the great majority of cases, is produced by the introduction of poisons into the body or by an auto-intoxication. The question of auto-intoxication is of great interest in this connection, for it is due to physiological error along one or other of two lines. It may be, and often is, due to physiological errors in primary digestion with the absorption of toxins from the gastro-intestinal tract. In other instances it is due to errors in the processes included under secondary digestion, such as retention of waste matters and imperfect katabolism. The latter occurs in the aged, in gouty and rheumatic people, and in the sedentary. The former is perhaps more common in young adult life. The condition is present, probably, in all diseases in which there is absorption of deleterious substances. It may even be caused by syphilis, and manifest itself, as in one of the cases I have referred to, before there is much permanent thickening of the vessel wall.

Connection between arterio-sclerosis and hypertonus.—This brings me to the final question as to the relationship between arterio-sclerosis and hypertonus. I connect them thus: recurring or continued hypertonus leads to hypertrophy of the muscular media of the arteries under the physiological law that increased action in muscle leads to its hypertrophy. I am not concerned at present to inquire how the increased action is brought about; I content myself by showing its existence. This accounts for the thickening media. The thickened intima is to be explained by the circulation in the blood of deleterious substances of various kinds; these substances act on the sub-endothelial connective tissue, irritate it, and lead to its hyperplasia. We thus get the completed picture of arterio-sclerosis. The evolution of the changes occupies a varying time in different persons; in some the hypertonus comes early in life.

and in early middle age sclerosis is established. In some there is little physiological error, hypertonus is but an occasional occurrence, and sclerosis appears late in life. The capability of the arteries to contract when an excess of toxin is present is in my experience seldom lost, and that is the reason why the recognition of it in sclerosed vessels is a matter of such great practical importance. While for the present I do not deal with the behaviour of the capillaries, it is not to be understood that I ignore their presence, their reactions, or their influence in some of the conditions touched upon in this paper.

Meeting VIII.—May 15th, 1901

Mr A. GORDON MILLER, *President, in the Chair*

I. EXHIBITION OF PATIENTS

1. *Dr Alexander Bruce* exhibited—

(*a*) A case of PARALYSIS OF RIGHT BRACHIAL PLEXUS, due to injury.

(*b*) A case of a very acute disseminated SCLEROSIS OF THE CEREBRO-SPINAL TYPE.

2. *Dr Byrom Bramwell* exhibited—

(*a*) A peculiar case of MUSCULAR ATROPHY. The patient, a sailor, aged 41, enjoyed good health until September 1898. After sleeping in the open air on a stormy night, after indulging too freely in beer, he felt sore all over, but was able to resume his work the next day. Ten days afterwards he began to suffer from cramps in the left leg and from weakness in the left foot, beginning in the big toe. The loss of power in the leg gradually increased. Two months afterwards the right leg became affected. Eight or nine months later the hands and forearms became involved. In May 1900 he was unable to stand or walk without support, and could not lift a cup to his mouth because of weakness in the hands and wrists. With the exception of pain due to "cramps" in the affected parts, there were no sensory symptoms. The bladder and rectum had not been affected. Since May 1900 he had improved, and was now able to walk considerable distances without support. The extensors on the front of the legs and

the muscles of the hands and forearms were still markedly atrophied. The progress of the disease seems to have been arrested, but for the last few months there had not been any marked improvement.

The case was a peculiar one. At first sight it might appear to be an ordinary case of progressive muscular atrophy of spinal origin, but the onset in the lower extremities, and the facts that the atrophy was confined to the peripheral parts of the limbs (legs and feet, forearms and hands) and that marked improvement had taken place, seemed opposed to this view. It did not appear to be a case of ordinary peripheral neuritis. Beri-beri had, of course, been thought of, but there was nothing to show that it was due to that condition. Some cases, which appeared to be similar in nature, had been described, in which the peripheral nerves had been enlarged; in this case, there was a difference of opinion as to the condition of the ulnar nerves—some observers thought they were enlarged, others were unable to satisfy themselves on this point. Dr Bramwell intended to publish a detailed account of the case and of the literature referring to it.

(*b*) A case of SYRINGOMYELIA in which there was very marked atrophy in the small muscles of the hand and forearm.

3. *Dr Edward Carmichael* exhibited a patient with a HYPERTROPHIC CONDITION OF INTEGUMENT, probably due to distended lymphatics.

D. L., age 57, compositor. Two conditions to be noticed.

(1) Skin and subcutaneous tissues.

(2) Right knee joint.

1. CUTANEOUS SYSTEM.—Twenty years ago he got a blow with a cricket ball on cheek, and after the wound healed a somewhat keloid condition appeared, and has gone on increasing. But so far as leg is concerned, the first appearance of swelling was last September (eight months ago), when he got an injury to knee, and after he began to try to walk the swelling appeared first over the calf, and extended down. Since his admission it has extended up over buttocks.

There are further several small nodules over body and other leg, one of which was excised and sent to the Laboratory, and showed the microscopic appearance of a fibro-sarcoma.

2. THE KNEE JOINT.—Six years ago he slipped on going down stairs, and felt something crack in his right knee. He walked home two miles, lay in bed for a week and fomented, after which he got to work, limping for about five weeks, and then to all appearance his knee was perfectly well.

Last September he slipped on the street, and his leg doubled under him. He rested and fomented again, and then tried to walk, but found the leg very useless, and since then it has gradually got worse.

The joint feels quite disorganised, you can get the fingers right between the articular surfaces, the ligaments seem gone, the external condyle atrophied, and on movement a grating is felt below patella.

It simulates a case of Charcot's Disease, but there are no very marked symptoms of spinal cord lesion.

Tactile Sensation—sensation to pain and heat and cold, all slightly less on right leg, but probably not more so than might be explained by considering the altered sensation from subcutaneous swelling.

Voluntary muscular movements are excellent.

Superficial Reflexes—plantar normal, or slightly greater than left; cremasteric present, but less marked than left; abdominal present, but less marked than left.

Deep Reflexes—Knee—rather abnormally marked on both legs, possibly more on right; ankle clonus absent.

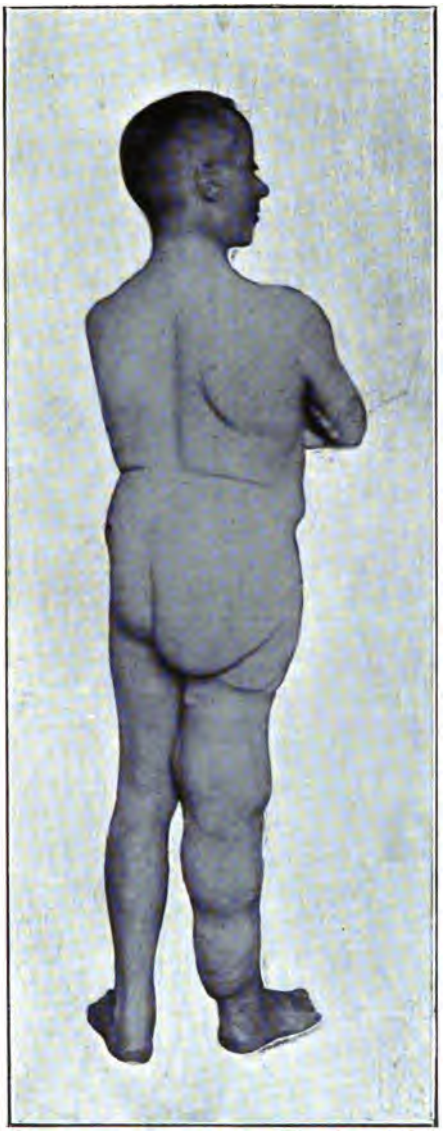
Organic Reflexes normal.

He has been a heavy smoker, a fairly heavy drinker, but has not had delirium nor syphilis, and has never been abroad.

The treatment we used was rest with elastic bandages, and of course iodide and arsenic.

The question of diagnosis is—Can we consider the whole condition of leg as due to a spinal cord lesion? Against this is the hypertrophied condition of cheek which commenced twenty years ago, and the very slight physical signs of a cord lesion, which are present. Or, then, have we to do here with a traumatism of knee, accompanied by some unusual subcutaneous condition—lymphatic, fibroid, or fatty—or of the nature of a sporadic elephantiasis?

4. *Mr F. M. Caird* exhibited two patients illustrating certain points in the diagnosis of GASTRIC CARCINOMA—



TO ILLUSTRATE DR EDWARD CARMICHAEL'S PAPER.

(*a*) A. G., æt. 48. Suffered from indigestion, vomiting, and emaciation. A large mobile tumour could be felt in the pyloric region. After a test meal hydrochloric acid found to be very scanty. Weight 7 st. 10 lb.

Laparotomy, 20th Dec. 1900.—The large tumour could not be removed, and a posterior gastro-jejunostomy was performed. Patient progressed satisfactorily, and now weighs 10 st., and not a trace can be found of the tumour, which probably was of a purely inflammatory nature.

(*b*) D. D., æt. 51. Indigestion, nausea, no vomiting, emaciation. Mobile epigastric tumour felt, the size of a small orange. Abundant free hydrochloric acid after test meal, also some lactic acid. Weight 8 st.

Laparotomy, April 1901.—Excision of pylorus and adjoining area of stomach. The free ends of the duodenum and stomach were then closed, since they could not be approximated, and a posterior gastro-jejunostomy was performed. Microscopic examination shows the trouble to be a typical columnar cell carcinoma, but the patient has progressed favourably, and daily gains weight so far.

5. *Mr Alexis Thomson* exhibited a patient after enterectomy for MULTIPLE TUBERCULOUS STRICTURE of the small intestine. The patient, a woman, aged 30, presented the features of chronic incomplete obstruction of the small intestine. She had had an attack of inflammation of the bowels nine years before, but had been quite well until her present illness, which began a month ago. On examination of the abdomen, there were a series of distended coils, showing occasional peristaltic movements in the region of the umbilicus, and between the coils a number of ill-defined indurations could be felt.

The obstruction was regarded as tuberculous in origin, and as probably depending upon stricture or matting of the bowels, or a combination of these lesions. The abdomen was opened on the 8th of March, exposing a number of greatly distended and hypertrophic coils of small intestine. These were partly matted to each other by connective tissue adhesions, and showed diffuse miliary tuberculosis of their peritoneal coat. The diseased portion of bowel was found to be the middle and lower thirds of the ileum, the upper portion of the ileum and

the jejunum being healthy. After liberating the affected coils from each other, a series of well-marked annular strictures were found all included within a portion of bowel less than two feet long.

The most distended loop of bowel was stitched to the edges of the wound and drained through a Paul's tube. Twelve days later, when the bowel had been completely emptied, the portion comprising the strictures was resected and the divided ends joined by sutures. She is now quite well, but requires to take a little castor oil every night to prevent her becoming constipated.

The portion of bowel removed measures 22 inches in length; it presents five annular fibrous strictures, none of which will admit the tip of the little finger; between the strictures the bowel is enormously distended and hypertrophied.

6. *Dr F. D. Boyd* exhibited a patient suffering from acquired PULMONARY STENOSIS. The murmur was characteristic. It was superficial, rough in tone, systolic in time. The point of differential maximum intensity was in the second left interspace to the left of the sternum. It was conducted upwards along the left border of the sternum. The area of audition was to the left of the sternum and upon the sternum. There was no cyanosis and no clubbing of the fingers.

7. *Mr H. J. Stiles* exhibited a boy, aged 12 years, whom, six months ago, he had trephined for CEREBRAL APOPLEXY with rupture into the right lateral ventricle. The patient, while enjoying good health, was seized, without any apparent cause, with a succession of general convulsions followed by unconsciousness and complete left-sided hemiplegia. The following day he was removed to the Children's Hospital. As no improvement had taken place at the end of five days, Dr Fowler, under whose care the patient was, asked Mr Stiles to see the case, with a view to surgical treatment. It was decided to explore the right hemisphere. On exposing the right motor area, the intracranial tension was found to be so great that the cerebral cortex, after protruding through the opening in the skull, ruptured spontaneously. On introducing the finger into the rent a quantity of blood-clot was felt occupying the right lateral ventricle and the adjacent basal ganglia. The

clots, which were removed with a lithotomy scoop, were dark in colour, and apparently a few days old. The following day the patient was quite conscious. There was at first a copious flow of cerebro-spinal fluid, which gradually ceased. A hernia cerebri also developed, but gradually disappeared. Two months after the operation the paralysis of the lower extremity had so far recovered that the patient was able to walk without difficulty. At the present time (six months after operation) there is no paralysis of the face or leg, but the forearm and hand have only partly recovered. At the operation it was thought that the lesion was a hæmorrhage into a glioma; the fact, however, that the boy is now in perfect health points to the lesion having been a simple apoplexy.

8. *Mr J. M. Cotterill* exhibited—

(*a*) A case of extensive rodent ulcer of the face. Four years ago Mr Cotterill had enucleated the eyeball and removed the orbital contents, malar bone, and part of the left superior maxilla. The patient had remained free of disease for about two years; but the ulcer having lately broken out afresh and being inoperable, she had been subjected to the X-Ray treatment. Improvement was marked, the ulcer was cicatrising, and pain was considerably less.

(*b*) A case of JOINT AFFECTION in a hæmophilic patient.

Three years ago the patient (a boy of 15) had presented himself with a swelling of the left knee-joint, closely simulating tuberculous disease. On closer examination, however, it proved to be hæmathros, and blood was removed from the joint by aspiration. Recovery had taken place in the knee, with perfect movement.

The condition had now affected the left elbow, and was interesting in the fact that it had been going on for some three years, sometimes being better and sometimes worse.

The case was interesting in its close resemblance to tubercular disease, and in the danger to life which there would be in operative interference.

II. EXHIBITION OF SPECIMENS

1. *Dr A. Scot Skirving* exhibited—

(*a*) BRAIN SHOWING COMPLETE TRACK OF MAUSER BULLET.

The bullet entered in the left frontal region and, having

almost completely ploughed up the right internal capsule, emerged in the right angular gyrus. It then turned at right angles to its former course, and tearing through the occipital cortex, finally lodged just posterior to the parieto-occipital fissure. The patient was trephined twice and lived 18 days, the immediate cause of death being lung complications. The brain remained quite free from sepsis. A full account with photographs will appear in the "Edinburgh Hospital Reports."

(*b*) BRAIN FROM CASE SIMULATING RUPTURED MIDDLE MENINGEAL ARTERY.

The patient, a woman of 53 years, had fallen down stairs and struck the right posterior temporal region producing a wound but no fracture. When the patient was seen some five hours after the accident, signs of an extensive basal fracture were obvious. In addition, however, the following were present —widely dilated left pupil and contracted right; general flaccid paralysis of the limbs, but supination of left forearm on pinching the skin; pulse full and 48 per minute. On account of these symptoms a disc of bone was removed over the meningeal area. No clot was found external to the dura which was tense and did not pulsate. It was therefore incised, when perfectly *fluid* blood, evidently under very high tension, gushed up at once out of the dural opening. On inserting a finger a cavity as large as a Tangierine orange was found to be present, a distance of rather more than 1½ inches intervening between the brain and the cranium at the deepest part of the depression. The blood was removed by swabs and the cavity lightly packed with gauze; on removal of this some twenty minutes later the surface of the brain was found to be only ½ inch from the bone and the pulse-rate had risen to 72. With this exception no improvement followed, and the patient died 24 hours later having remained unconscious since the accident. Post-mortem examination showed little or no further hæmorrhage and a fracture right across the base, involving both temporal bones. There was also extensive laceration (apparently from centre cusp) of the adjacent portions of the temporo-sphenoidal and lower frontal lobes. No blood whatever was found at the base of the brain, the blood having apparently therefore made its way upwards. The brain had expanded very little if at all, the maximum of "recovery" having apparently been reached not long after the operation.

(*c*) STOMACH OF A PATIENT EIGHT DAYS AFTER OPERATION FOR PERFORATED GASTRIC ULCER.

The patient was a girl of 19 years. The history, onset and symptoms were typical. Laparotomy was performed about three hours later. A prolonged and careful search for the opening failed to find it at first owing to its minute size. An incision was then made laterally and the duodenum examined with a negative result. Renewed examination of the posterior wall of the stomach disclosed an opening which did not much more than admit a probe. This was closed by a double row of Lembert's sutures. The abdomen was flushed out and drained by the lateral wound. The case did well for four days, but died of peritonitis on the eighth. The sutured stomach was, however, found perfectly watertight.

(*d*) SPECIMENS ILLUSTRATING BULLET IMPACT.

(*a*) TWISTED THIMBLE OR SHEATH OF MAUSER BULLET. —This with small portions of saddle and saddle cloth were removed from a pelvic wound in which the course of the bullet had been remarkable. First piercing the horse and then the saddle it had entered the rider (a yeoman) in the middle line between the anus and coccyx. The finger passed into the track easily felt a rough and bare area on the posterior surface of the pubis. No blood appeared in the urine nor fæces in the wound, and the patient made an excellent if somewhat tedious recovery.

(*b*) FRAGMENTS OF FEMUR.—There were some 15 or 16 pieces removed from the thigh of a Boer on whom amputation had later to be done. One fragment (three inches in length) showed on one side the distinct neat groove of the bullet. The specimens illustrated the shattering effect an ordinary non-expanding bullet may not infrequently produce.

(*e*) LARGE PERINEAL TUMOUR (with photographs).

This, a lipoma, resembled a large melon in size, and weighed 5 lb. The patient from whom it was removed was under Dr P. H. Maclaren's care, and was 80 years of age. He stated that the tumour had grown in 12 years. It was attached by a stout short pedicle just posterior to the anus, and hung down between his legs, making progression and defæcation alike a matter of no little difficulty. The tumour was removed by Mr Shaw M'Laren with an excellent result.

2. *Dr Alexander Bruce* exhibited photographs and microscopic specimens illustrating PROGRESSIVE MUSCULAR ATROPHY, AMYOTROPHIC LATERAL SCLEROSIS, and GLIOMA OF THE PONS VAROLII.

3. *Mr F. M. Caird* exhibited—

(*a*) Two vesical calculi removed from a gentleman about 68 years of age, who also suffered from enlarged prostate. Suprapubic cystotomy was performed, the stones removed, the prostatic canal stretched with the finger, and the bladder sutured. Union by first intention.

(*b*) A large simple papilloma removed by suprapubic cystotomy one year ago from the bladder of a patient aged 65. Suture. Union by the first intention.

(*c*) Two similar smaller growths from the same case removed this year. Suture. Primary union. The cases required a very limited use of the catheter to draw off the water after operation.

(*d*) A large vesical calculus weighing about half-a-pound. In this case Mr Cathcart's system of syphon drainage proved a great comfort to the patient, who made a good recovery.

4. *Mr Alexis Thomson* exhibited—

A GLASS-BARRELLED SYRINGE provided with nozzles suitable for irrigating the urethra or bladder or auditory meatus, and with trocars suitable for aspirating collections of fluid, or for injecting iodoform emulsion into abscesses or joint cavities.

5. *Dr Logan Turner* exhibited—

(*a*) Two casts showing congenital malformation of the auricle.

(*b*) A larynx from a case of primary laryngeal diphtheria.

6. *Mr H. J. Stiles* exhibited—

(*a*) Intestines from a girl, aged 2½ years, who had suffered from the condition known as idiopathic distension of the colon. The whole of the large intestine was greatly dilated, and its wall considerably hypertrophied. Three inches above the anus the rectum became suddenly narrowed to the diameter of the little finger. The narrowing extended upwards for two inches to form a tubular constriction, which only just admitted an

ordinary lead pencil. The wall of the constricted portion was about three times the thickness of the rest of the large intestine; its mucous surface was thrown into longitudinal folds, that of the bowel above being perfectly smooth. The child had suffered from constipation ever since birth; its abdomen was habitually distended, and the peristaltic movements of the intestines were generally visible. The small intestine was practically normal. It was evident that the so-called idiopathic dilatation of the colon was in reality secondary to a congenital narrowing and hypertrophy of a portion of the rectum, a state of affairs which presents a close analogy to congenital hypertrophy of the pylorus. No doubt both conditions are essentially dependent upon an error in development. Mr Stiles referred to an infant upon whom he had recently operated, and found that the whole of the large intestine was of the diameter of an ordinary penholder; the wall was greatly thickened, and the lumen would barely admit a small probe.

(*b*) A sarcoma of the right testicle removed from an infant aged nine months. The testicle was first noticed to be enlarged when the child was three months old. During the last month growth had been more rapid, and at the time of operation the testicle was the size of a small hen's egg. The organ was heavy, firm, smooth, and almost uniform in consistence; the cord was a little thickened. No glandular enlargement could be discovered. The child was otherwise strong and healthy. The microscope revealed a typical spindle-celled sarcoma.

Meeting IX.—June 5th, 1901

MR A. G. MILLER, *President, in the Chair*

I. ELECTION OF MEMBER

J. W. Leech, M.D., Newcastle, was elected an Ordinary Member of the Society.

II. EXHIBITION OF PATIENTS

1. *Dr R. Purves* exhibited a case of MUSCULO SPIRAL PARALYSIS after operation.

H. K., age 5 years. Fall on arm on 11th May 1900,

resulting in sep. of l. epiph. of humerus, which united with some displacement. In end of August was seen at Deaconess Hospital with drop wrist, some power of supination left. *8th September*, nerve exposed in lower third of arm, followed down was found to dip into the hollow beneath the projecting edge of the diaphyses, and was firmly adherent to the bone, was released, and displaced to one side. Under massage and electricity, he was able to straighten wrist in December, and by Christmas was almost recovered.

2. *Dr M'Kenzie Johnston* exhibited a patient, after the removal of LARYNGEAL PAPILLOMA at the age of 4½ years. The patient was sent to him suffering from loss of voice and dyspnœa, which at night was often very alarming. Examination with the laryngoscope made it clear that the obstruction was due to papillomata, which reduced the glottic breathing space to less than one-third of the normal. Repeated attempts at removal failed, because the various instruments tried were too large for easy introduction through the mouth. He had a special forceps made, with which the growths were removed with little difficulty, under cocaine, as the boy proved a model of pluck and steadiness. It is over six months since the operation, and there has been no signs of recurrence. The breathing is perfect, and though the voice is not as good as could be wished, this seems due to a minute tag lying anteriorly between the cords which the forceps has failed to catch.

3. *Dr Norman Walker* exhibited—

(*a*) A case of RINGWORM, which was the first which he had treated by X-rays. The boy had had the disease for about two years, during most of which time he had been under treatment, always with some benefit. The disease, however, after being apparently cured, recurred again and again, and Aldersmith's method of treatment was tried as a last resource, the boy having a presentation to one of the orphan schools. The method was apparently successful, and the boy went to school and remained there for some months. The disease, however, again broke out, and as it was limited to a patch about two inches in diameter on the vertex, Dr Walker suggested to his mother that the X-rays might be tried, explaining the

experimental nature of the treatment. The boy was exposed for ten minutes on ten successive days. There was never any sign of reaction, and at the end of this time the hair could be lifted out in handfuls, and in the course of a week was absolutely bald over the exposed area. Antiseptic ointments were applied, and apparently a cure had resulted in about three weeks. He did not suggest that this method of treatment was capable of wide application. Ringworm was so diffuse, that the whole head would require to be depilated, and to many this would appear an unduly severe method of treatment. At the same time, where a rapid cure was urgently necessary, as in this case, there was a good deal to be said for the treatment. He had allowed the boy to return to school.

(*b*) Several cases of ALOPECIA AREATA of special interest.

He had for some time been making a special study of this disease, and analysing the cases, bearing in mind the different theories of their development. The cases shown, each contributed something of interest, and they supported different theories of the disease.

The first case was that of a man, aged 47, who noticed, two years ago, that his hair had become white over round patches, scattered irregularly over his scalp. This condition remained without change until six or eight months ago, when patches of typical Alopecia Areata developed with complete loss of hair. He had no nerve symptoms, he was a strong robust man, apparently altogether without nerves, but undoubtedly the case would be claimed by those who supported the nerve theory of the disease.

Second, two brothers, one of whom was suffering from his second attack of Alopecia Areata, the first having occurred five years ago, and having completely recovered. The present attack began two years ago, and is now complete. His younger brother developed the disease two months ago, and now presents it in a very typical form.

Lastly, a case of complete Alopecia Areata in a corporal of the Scots Greys, of exceptionally robust physique. His person was almost entirely denuded of hair, and he had upon the bald scalp enormous *perfectly typical comedones*, along with considerable oily seborrhœa; this case appears to support Saboraud's theory that Acne and Alopecia Areata are closely allied diseases.

III. Exhibition of Instrument

Dr M'Kenzie Johnston exhibited Laryngeal Forceps for removal of growths in young children. The forceps was of the Dundas Grant pattern, made on a small scale, and made with a laryngeal curve instead of being bent at almost a right angle as in the original instrument. This makes them easier of introduction into the larynx.

IV. Demonstration

Dr G. L. Gulland gave a microscopical demonstration of specimens of Actinomyces Fungus prepared in various ways. The specimens were derived from a case of Dr Thin's. The patient had an abscess near the temporo-maxillary joint, and the pus contained in it was found to be full of Actinomyces granules.

V. Original Communications

1. OPERATIVE INTERFERENCE IN CASES OF STRICTURE OF THE LARGE INTESTINE

By F. M. Caird, F.R.C.S.Ed., Senior Assistant Surgeon, Royal Infirmary, Edinburgh

This paper may be regarded as supplementary to that read at the Society in 1895 (see vol. xiv. of the "Transactions of the Medico-Chirurgical Society of Edinburgh"), and is intended to give the results of extended experience and the after-history of the cases formerly narrated.

We may with some advantage generalise on a few preliminary points of interest. The diagnosis of stricture of the large intestine has rarely presented difficulties. The symptoms observed were mainly irregular action of the bowel, the presence of localised tympanites, the palpation of a somewhat mobile nodulated swelling, often tender on pressure, and occasional rises of temperature, due to septic absorption. The febrile attacks simulate those of influenza, and patients sometimes date their symptoms from a supposed attack of influenza. Occasional errors in diagnosis have arisen: thus acute appendicitis has been found when carcinoma was expected; a case of carcinoma has been recommended as appendicitis; and in a case where diagnosis was doubtful, an unsuspected

ectopic gestation was revealed. Again it is not always easy to discriminate between immobile neoplasms of the ascending colon, of the kidney or of the retroperitoneal tissue.

The mode of operation remains much the same as described in the former paper. After the usual purification of parts prior to incision, active antiseptics are discarded and warm sterile salt solution and sterile gauze swabs are alone employed, except in the case of special applications to the cut intestinal surfaces. Access is generally gained by incision in the left linea semilunaris directly into the peritoneal cavity. When necessary, more room can be obtained by a second incision carried outwards at right angles. The tumour can now be palpated and its relations established. Adherent omentum is severed and ligatured. The mass is withdrawn from the abdomen and packed round with gauze so that all subsequent procedure may be conducted externally to the shut off peritoneal cavity, and any direct interference with the septic interior of the intestine is postponed.

If the gut cannot be withdrawn the mesenteric vessels and adhesions should be divided *in situ* between double ligatures or between pressure forceps, and the free loop of bowel with the tumour can now be pulled out. It is advisable to divide the mesenteries well away from the gut so as to remove as much lymphatic tissue as possible. This means that large vessels have to be cut, and hence the greatest care should be taken in ligaturing firmly. Forceps must be trustworthy, and each mass of tissue and veins should be secured carefully *seriatim*. If the gut cannot be readily pulled out because of short mesenteries and adhesions, a couple of intestinal clamp forceps should be applied to the healthy freed colon well beyond the distal end of the tumour, a quarter of an inch apart. The bayonet-shaped clamps introduced by Conrad Brunner are excellent for this purpose, when improved by a peg to prevent the blades crossing. The gut is now divided between the clamps, whereupon the cut intestinal surfaces in the grasp of the clamps should be turned up and disinfected. This is done by rubbing a little pure carbolic acid over them, drying it off with gauze and smearing on some iodoform. The clamps and contained gut are next meanwhile wrapped up in gauze to obviate every chance of sepsis. The free end of the bowel is then pulled out and the various

adhesions, mesenteries, and vessels are successively ligatured and divided, as one works round the tumour, which is thus gradually freed from its surroundings until healthy colon or ileum is reached and severed between a second pair of clamps, and removed along with the tumour.

The proximal end of the gut in the clamp is of course treated similarly to the distal end and at once enveloped in gauze. Any enlarged lymphatic glands should be removed. The divided ends of the gut are now exposed, still in the grasp of the clamps. They should be approximated to note if the cut sections correspond in size and can readily be brought together. As a rule, owing to the marked distention and hypertrophy above the stricture, and to the atrophy beyond, there is less disparity than one would expect; or again the difficulty may be to accommodate the huge ileum to the somewhat atrophied colon. Fresh gauze is once more packed around the gut, the clamps are removed, and one may with advantage trim off part of the compressed intestine. There is but little bleeding. Should the cut sections differ widely in calibre, the smaller may be stretched with dressing forceps; or the two ends may be united after the manner employed to join stomach and duodenum in pylorectomy. End to end suture is made use of much as formerly. Three provisional stay sutures are introduced which pass through the whole thickness of the gut from the peritoneal aspect of the one section and back from the mucous surface outward to the peritoneal aspect of the other. The first and most important stay should be introduced at the mesenteric margin of the bowel and the other two equi-distant from it, so that the circumference of the gut is divided into thirds. The two extremities of the first stay knot are preserved: one lies towards the anterior aspect of the bowel, the other is pulled through the mesenteric gap and thus lies posterior to the gut. It is convenient to cut one extremity of the remaining two stay knots short, thus leaving one long free end. The end of the second suture and the anterior end of the first is held tense, and a continuous suture is now carried through the whole thickness of the gut from the first to the second stay knot. The second and third suture ends are now held tense and the suture continued to close the second segment of the circle, after which there only remains to make the third segment tense, which is now done by pulling on the

third stay knot and the posterior end of the first one. The bowel is thus rotated, and the continuous suture travels on and completes the union. A continuous Lembert suture is next carried all the way round, completely covering in the former deep sutures, and the stay knots once more come into play. All the sutures are now cut short. Additional Lembert sutures are inserted over the site of the stay knots and at any other point where it is deemed necessary, for example, in the vicinity of the mesentery. We have generally made the so-called blanket, button-hole, or sailmaker stitch, and have used the finest intestinal silk and the somewhat stouter No. 1 Chinese twist for stay knots. As a rule the wound may now be closed after the routine fashion, the use of the iodoform gauze tampon or the drainage tube depending upon the special features of individual cases.

A.—*Primary Resection and Suture for Malignant Stricture of Ileo-Cæcal Valve or Ascending Colon.*

1.[1] Miss K., æt. 47. Ileo-cæcal carcinoma. Nine inches removed, including portion of ileum, 1893. The tumour was a mass of columnar celled carcinoma about the size of a golf ball. (Pl. III. Fig. 3.) Wound closed without drain. Flatus third day. Healed well. Patient enjoys good health now.

2. Mrs P., æt. 40. Stricture of ascending colon. Action of bowels very irregular; repeated attacks of threatened obstruction. Excision of growth, including 2 inches of ileum and cæcum; in all, 10 inches; February 1897. The tumour, carcinoma, formed a solid ring-like structure 1½ inches long, traversed by a central channel which barely admitted an ordinary lead pencil. A mass of adherent omentum had also to be removed. No drainage. Patient made an excellent recovery. The prognosis was, however, bad, since on microscopic examination a small nest of cancer cells were found remote in a portion of the removed mesocolon, and the patient succumbed, as was to be expected, a year after from secondary growths in the liver.

3. Mr W. M., æt. 67. Very similar in site to last case, but the intestine was adherent to the abdominal wall. Ten

[1] See vol. xiv. "Trans. Med. Chir. Soc. of Edinburgh."

inches of bowel from the ileum onwards were removed, as also a considerable area of parietal peritoneum and transversalis muscle, on November 11th, 1896. (Pl. I. Fig. 2.) Much difficulty was experienced in uniting the colon and ileum. Wound drained with tube and iodoform gauze. A fæcal fistula formed, and recovery was somewhat slow. The patient was doing well in his own home when he was carried off by an epidemic of influenza on 10th January 1897.

4. Mr R. C., æt. 28. The patient had a hard firm mass in the appendix region which was removed by division of the ileum and the ascending colon and separation of the various adhesions. A pair of forceps slipped from off a large vein in the mesocolon and deluged the wound with blood, but the vessel was speedily secured. End to end suture. No drain. The perforation proved to be an early carcinoma of the tip of the appendix, which structure was reflected upon and adherent to the posterior aspect of the cæcum and projected as a hard mass into the lumen of the gut. (Pl. I. Figs. 3 and 4.) Operation, August 1896. Healed well. Patient has enjoyed good health since.

5. Mrs M., æt. 40. Patient has a large nodular hard mass to right of umbilicus filling up the lumbar region; painful on exertion, tender, rather fixed, and of three months' duration. There is no marked irregularity of the bowels, but she experiences some difficulty in micturition. Operation 23rd April 1901. Incision in linea semilunaris and at right angles to it. Tumour separated from liver, duodenum and omentum; removed with large piece of mesocolon. Kidney freely exposed and stripped. End to end union of ileum to hepatic flexure of colon. Drainage. The patient became restless and sick. On the following day the stomach was washed out on account of continuous vomiting of coffee ground character. She sank on April 25th. T. 103, P. 140, R. 44. The sectio showed congestion of both kidneys; old and recent endocarditis, antecedent to the operation; a peculiar swollen condition of the pancreas and marked *post-mortem* digestion of the stomach and œsophagus.

6. Mr A. C., æt. 32. Supposed to be suffering from appendicitis, having suffered from localised pain and vomiting two years ago and again five weeks ago. Formerly constipated, the bowels now act well without medicine. There is a mobile

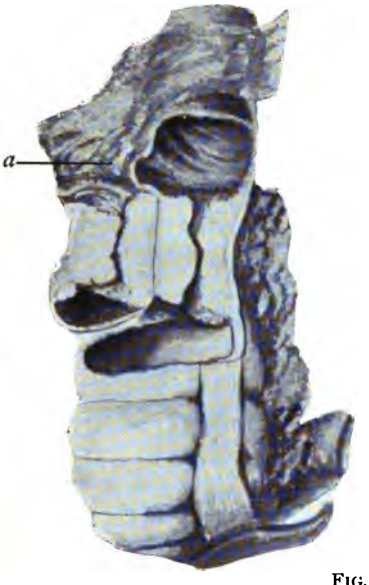


FIG. 1.

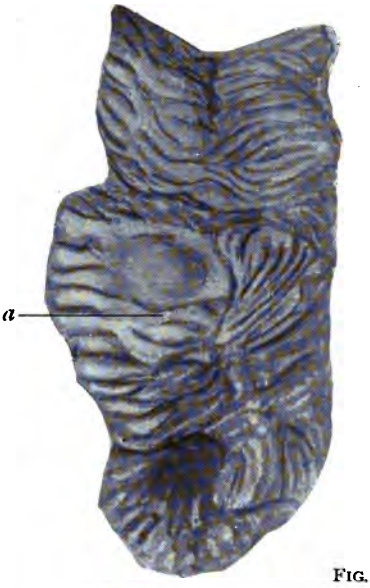


FIG. 3.

FIG. 2.

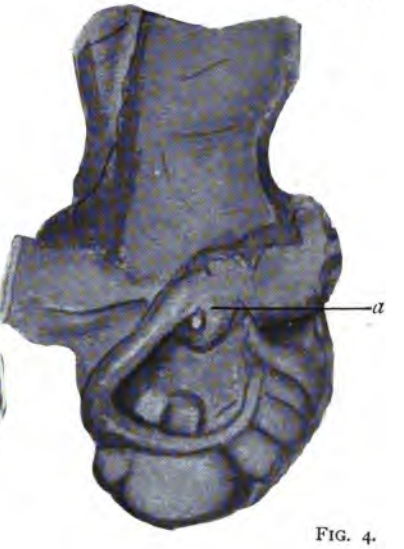


FIG. 4.

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TO ILLUSTRATE MR CAIRD'S PAPER.

FIG. 1.—Case 2. Malignant stricture of ascending colon. Parts removed at operation. The strictured area divided longitudinally and turned back to show the narrowed channel. Adherent omentum, *a*.

FIG. 2.—Case 3. Cæcum laid open to show ulcerating malignant stricture.

FIG. 3.—Case 4. Cæcum laid open, projecting carcinomatous nodule corresponding to adherent tip of appendix *a*.

FIG. 4.—From same preparation. Back view, tip of appendix *a*.

globular swelling about the size of an orange in line with the highest point of the iliac crest internal to the anterior superior spine. Immediately internal to this dull area was a resonant tense swelling which dispersed under pressure and was correctly thought to be flatus behind an ileo-cæcal stricture. Operation, 10th April 1901. A dense tight annular stricture of the ileo-cæcal valve found. (Pl. III. Fig. 2.) The ileum was hypertrophied to the dimensions of the great gut, and the colon was small and thin. End to end suture. No drain. Flatus third day. Healed well. Home 4th May.

B.—*Resection. Artificial Anus. Incomplete Operation.*

7. Mrs C., æt. 59. Three years ago noticed a small swelling about the region of the appendix: she had then pain in the thigh, frequency of micturition and pain before the act. This was followed in eighteen months by diarrhœa which has since persisted, and she is much thinner. There is now a hard firm mass in the region of the ascending colon as large as a couple of fists and rather fixed. Operation, December 1901. Gut divided at ileum and hepatic flexure of colon, a mass of omentum and mesocolon clamped and cut, and the huge tumour was turned outwards. It was then found to be adherent to the kidney and to the duodenum. Part of the kidney and a small portion of the duodenum had to be removed along with the growth. There was troublesome venous hæmorrhage which was thought to be satisfactorily arrested. It was decided to tie a couple of Paul's tubes into the divided colon and ileum, and at a later date to close the artificial anus. The patient had to be transported a considerable distance and to be carried in a lift to her ward. On arrival there she was found moribund from internal hæmorrhage. The sectio revealed a rent in a large tributary of the portal vein which had not been secured.

C.—*Lateral Anastomosis for Ileo-Cæcal Tumour.*

8. Mr G. F., æt. 35, five weeks ago felt a sharp shooting pain in the right groin, accompanied by nausea. This passed off in three or four hours, and patient took to bed for one day. He resumed work, but pain has continued ever since, increased on exertion, keeping him from sleeping, and extending down

the front of the thigh and sometimes through to his back. He cannot lie on the left side, and has a dragging sensation on the right side. There is a hard, oval, tender swelling in the right groin. The bowels are regular. He has lost weight during the last five weeks. Operation, 6th March 1901. A large mass, the size of the fist, firm and nodular, was found in the inner aspect of the psoas muscle. It lay over the vessels adherent to them, and to the anterior crural nerve, and was incorporated with the ileum close up to the ileo-cæcal valve. Removal being out of the question, a coil of ileum above the tumour was laid alongside the upper part of the cæcum, and a lateral anastomosis effected. Flatus passed the following day. Suppuration of wound, 14th May. Left hospital June 11th.

D.—*Primary Resection and Suture for Innocent Ileo-Cæcal Stricture.*

9. M. S., æt. 9. An emaciated boy with constant diarrhœa, "ladder" patterns, and tumid abdomen; tender ileo-cæcal region. Removal of stricture with part of ileum and colon, 1899. End to end suture. No drain. Patient well, 1900.

E.—*Resection and Secondary Suture for Innocent Ileo-Cæcal Stricture.*

10.[1] Nellie S., æt. 7. Similar to above case. Excision of papillomatous stricture and adjacent gut (Pl. III. Fig. 1), 1st June 1895. End to end suture, 5th June 1895. Patient well, 1900.

F.—*Carcinoma of the Sigmoid Flexure. Primary Resection and Suture.*

11. Mr G. S., æt. 36. Removal of carcinoma of sigmoid, freely mobile, size of fist, 5th August 1896. No drainage. Perfect recovery, death from secondary growths in liver, February 1897.

12. Mrs McK., æt. 58. A healthy-looking woman who began to suffer in February from a constant dull aching pain in the region of the left side of the abdomen, together with

[1] See vol. xiv. "Trans. Med. Chir. Soc. of Edinburgh."

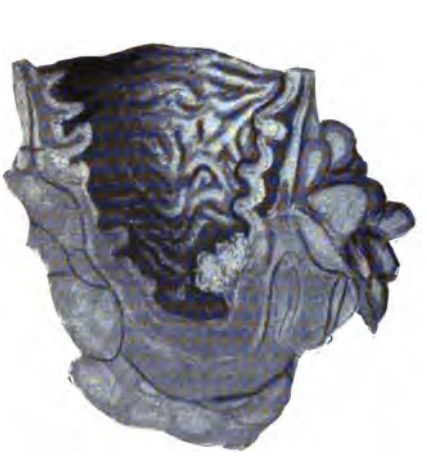

FIG. 1.

FIG. 3.

FIG. 2.

FIG. 4.

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FIGS. 1 and 2.—Case 12. Carcinoma of sigmoid flexure. Shows dilatation above the stricture. The intermediate segment, half an inch in thickness, which barely admitted a bristle, is not represented.

FIG. 3.—From the same case. Shows gangrene of the bowel extending above and below the intact line of sutures. Rupture has taken place close to the line of demarcation.

FIG. 4.—Case 15. Typical ulcerating carcinoma of the sigmoid flexure.

frequent paroxysms of more acute nature. There has been more or less constipation, the accumulation and passage of "wind" and of blood-stained mucus. She complains of increasing weakness and loss of weight. At the same time a lump was felt in the sigmoid region. It was plainly visible in May, and now is palpable as an irregular elastic fixed mass about the size of one's fist.

Primary resection and suture, 26th September 1898. The tumour, a large villous looking columnar carcinoma (Pl. II. Figs. 1 and 2), was firmly adherent to the peritoneum over the psoas and ilium, and its removal entailed the exposure of a large extent of retroperitoneal cellular tissue. A considerable amount of mesocolon had also to be taken away, but the vascular supply of the gut was good, the trimmed margins bleeding freely prior to suture. The hugely dilated oval extremity had to be partially closed before its lumen coincided with that of the small contracted distal extremity.

The patient progressed favourably and had a motion on the fifth day, and was passing flatus freely till 2nd October, when flatus ceased, she had pain over the cæcal region, and in the afternoon began to vomit a little. On the following day the pulse and respiration quickened, the abdomen moved well in respiration, no flatus passed, but fæcal matter was removed by enemata. A drainage tube was inserted into the wound and fœtid pus escaped. Next day she rapidly began to sink, free fæces passed from the wound, which was now opened up and washed out, and the patient died in the evening, 5th October.

At the sectio it was found that the gut was gangrenous over an area of about 2 inches. The gangrene began 1 inch *above* the level of the sutures, and here two or three large perforations ran into one as the parts were removed. The line of stitches remained intact. (Pl. II. Fig. 3.) The fæcal matter lay in the vicinity of the rupture, shut off by adhesions from the general peritoneal cavity. It is worthy of note that the patient had difficulty of micturition, and that the catheter had to be used from 1st October onwards.

13. Mr S., æt. 38. Observed a rounded mobile swelling in the sigmoid region in January 1900. He had some irregularity of the bowels, and the tumour increased, while he

lost strength and weight. Resection, end to end suture, June 1900. The tumour was firmly adherent to surrounding coils of gut and peritoneum, and with difficulty was removed under peculiarly disadvantageous circumstances. Drainage. Patient at present strong and well.

G.—*Carcinoma of the Sigmoid Flexure. Resection and Secondary Suture.*

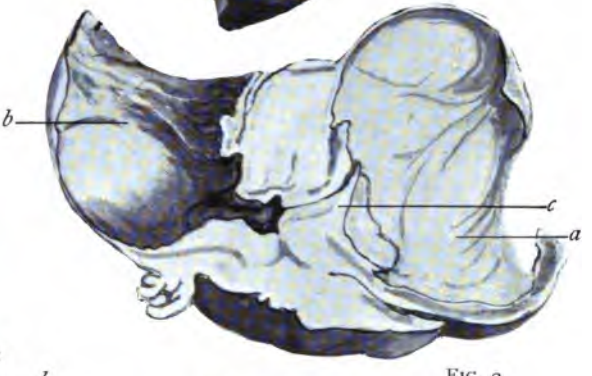
14.[1] Mr D., æt. 60. Anterior colostomy for obstruction, 19th March 1895, followed by resection of gut and end to end suture, 9th April. Patient made a good recovery, gained weight, and was in excellent health when, ten months after, sudden obstruction again developed. A second colostomy only gave temporary relief. No tumour, only adhesions discovered, and he died 17th January 1896. Permission for an autopsy was refused.

15. Mrs T., æt. 65. Patient suffered from obstruction, for which colosomy was performed at a period when she was already very weak. The bowel was acting freely through the artificial anus, but the patient showed signs of sinking from septic absorption, and yet had no peritonitis. Fearing that this might be due to continuous absorption from the ulcerated surface of the typical carcinomatous stricture (Pl. II. Fig. 4) which had caused the obstruction, the tumour was pulled forth on the second day, removed, and end to end suture applied. The patient only survived a few hours.

16. Mrs J., æt. 47. A uniformly healthy woman, who was suddenly seized with vomiting and obstruction. The vomiting ceased three days ago, but there has been no passage of flatus for eight days. She is greatly distended, but suffers little. Anterior colostomy was performed 5th September. An annular sigmoid stricture could be felt. Resection and suture, 8th September. Drain and iodoform tampon. Patient had a good night and passed flatus, but that evening became delirious, and the pulse rose to 120; the respirations were not quickened, and the abdominal movements were perfect. Transfusion was carried out with temporary benefit. The patient died 10th September, at 5.30.

[1] See vol. xiv. "Trans. Med. Chir. Soc. of Edinburgh."

FIG. 3.



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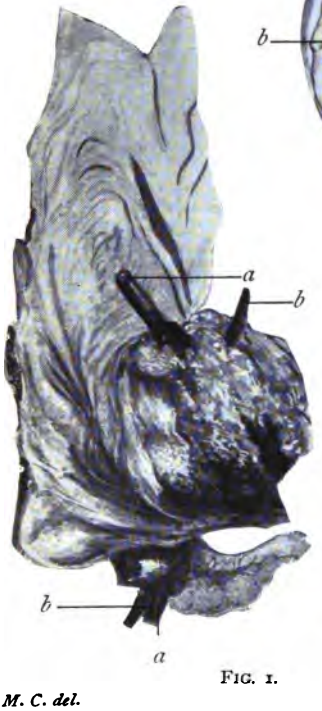

FIG. 1.

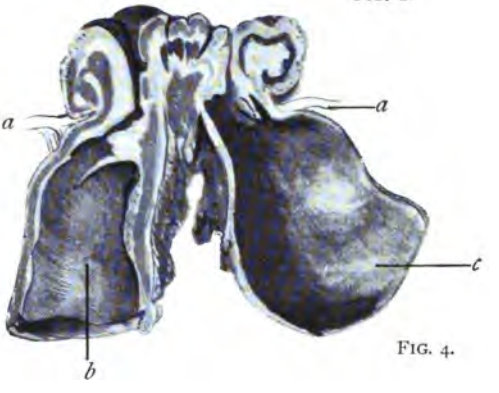

FIG. 4.

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FIG. 1.—Case 1. Cæcum laid open shows fungating carcinomatous mass. This was traversed by two narrow channels into which drainage tubes have been passed from the ileum onwards, *a* and *b*.

FIG. 2.—Case 6. Ileo-cæcal region in longitudinal section. Distended small intestine *a*, rather atrophied large intestine *b*, very narrow communication *c*, through the midst of a dense stricture.

FIG. 3.—Case 10. Simple papillomatous ileo-cæcal stricture. Hypertrophied ileum *a*, atrophied cæcum and large intestine *b*.

FIG. 4.—Case 19. Artificial anus in longitudinal section. Level of skin *a*. Functioning oral end of gut *b*; note hypertrophy. Flaccid disused distal gut with atrophied walls *c*.

Sectio. Not a trace of peritonitis; wound perfect; no leakage; nothing in pouch of Douglas, save a little serum; kidneys healthy. Death from cardiac failure.

17. Nellie B., æt. 39. Three weeks ago had severe attack of pain, constipation, and vomiting; the conditions on admission persists to a minor extent. She had a similar attack two years ago, but in other respects has been strong and healthy. A mesial exploratory incision was made below the umbilicus and an annular sigmoid stricture found, which was now projected through the left linea semilunaris and the mesial wound closed. Resection and formation of artificial anus was carried out 3rd October 1900. Secondary end to end suture, 15th October. Drainage. Iodoform tampon. Small fæcal fistula, 25th October. This was healed in November. Patient now strong and well.

18. Mr J., æt 55, had anterior colostomy performed in December 1898, for obstruction due to an annular carcinoma of the sigmoid. It was intended to perform resection, but the patient left hospital and did not return till February 1901. He seems to have enjoyed good health of late, although the liver is somewhat enlarged. On introducing the finger into the distal aperture of the artificial anus the tumour was reached. Resection of the artificial anus and tumour with end to end suture, 15th February 1901. It was thought wise to drain the wound since there had been so much peritoneum stripped from off the iliac fossa in removing the tumour. Bowels acted two days after, the first action by the natural passage for two years. Stitches removed and healed, 10th March.

19. Mr A. M., æt. 56. About a year ago he suffered from constipation and passed some blood. There was no diarrhœa. For the last five or six months he has had violent pains in the left inguinal region, aggravated by aperients. There is a hard mass to the left of the middle line in the basis of the pelvis. Resection of the tumour and formation of artificial anus, January 30th, 1901. The growth was found fixed to the pelvic brim and its removal entailed much stripping of peritoneum. Resection of the artificial anus (Pl. III. Fig. 4), 20th March. The operation was prolonged and difficult from adhesions, and the wound was packed with an iodoform tampon: union was not perfect and a small fæcal fistula formed. For the closure of this he

returned to hospital, and after unsuccessful attempts had been made he sank exhausted.

The sectio disclosed numerous large secondary growths in the liver.

H.—*Carcinoma of the Sigmoid Flexure. Resection. Artificial Anus. Incomplete Operation.*

20. Miss B., æt. 35. Suffering from seven days' obstruction, with, however, slight passage of flatus, vomiting, distended abdomen and ladder pattern. The cæcum very tender, nothing to be palpated, but it was stated that one tender spot in the left iliac region had been detected a couple of days ago. Her symptoms dated from five months back, when she was seized with pain in the cæcal region, since continuous, followed latterly by six weeks' constipation. Operation, 30th July. Incision over tender area through right linea semilunaris. Distended colon found, and the hand carried to the sigmoid flexure found it also distended above and below a typical annular malignant stricture. An incision was therefore made over this on the left side, and the tumour and loop of bowel pulled out. The right wound was then closed. The loop of bowel and tumour were then removed and a couple of Paul's tubes tied in. The artificial anus acted well, but the patient's pulse rose in frequency, she became delirious, and after lying unconscious for a couple of days, died on August 4th. There was no evidence of peritonitis or sepsis.

As far as the above cases go we may note that carcinoma affects the sexes in the same proportion, and that contrary to the experience of some others, the operative mortality is greater among women. Moreover our death-rate following resection and suture appears greater than it really is as far as operation is concerned, since three of the patients succumbed to auto-intoxication, the result of obstruction of the bowels, just as may happen after any colostomy. Hence it is a matter of regret that we attempted the union which was carried out in cases 15 and 16, although the result would still have been similar to that of number 20, where the patient died despite the surgeon's intervention. The fatal case 5 (cardiac failure) was due to our not recognising the bad general state of health which should

have contra-indicated operation. The death from hæmorrhage was a calamity also which should have been avoided. The bleeding vessels had been carefully sought and secured; we were fully satisfied that not one had been missed. The probability is that the twisting of a coil of gut restrained the bleeding while we were closing the duodenum, and that as the bowel returned to its normal position during the transit of the patient downstairs the venous rush took place. The gangrene of the intestine (12) is of great interest. It may be explained by some vascular thrombosis or kinking of the vessels during peristalsis, since it occurred late; the patient had been progressing well, and at the conclusion of the operation care was taken to ascertain that the parts were vascular. The case serves to support strongly the advantages of the Mikulicz iodoform tampon, and of the special method he has introduced for removing intestinal tumours. This is to withdraw the tumour and loop of bowel and leave it dependent from the wound, shutting off the peritoneal cavity. At the end of 36 to 72 hours, adhesions have formed and the mass may be removed, leaving an artificial anus, which is treated in about 14 days after with the special forceps introduced by Mikulicz for the spur, and the resulting fæcal fistula is next dealt with. In 12 cases so treated there was no instance of infective peritonitis.[1]

At present, experience leads one to believe that where there is *no obstruction*, the best treatment in stricture of the large intestine is primary resection, end to end suture and closure of the abdominal wound without drainage. If it be found true that the mortality rises to about 50 per cent., and that infective peritonitis be the chief factor,[1] then the procedure of Mikulicz will be welcome indeed.

As a rule two stage operations should be avoided. But they are to be rigidly adopted in presence of *an existing obstruction*, every trace of which, as is evident from our mishaps, must be got rid of before one thinks of attempting union. So also in weak patients or when the primary resection taxes the resources of the patient too greatly. In such cases we have left a more or less open wound and employed the iodoform tampon, a method of treatment also required when there is any doubt as to the vascularity of the bowel or when there has been much stripping of the peritoneum. The secondary

[1] "Handbuch der Deutschen Chirurgie," p. 465.

operation is however always more difficult than the primary. The presence of an artificial anus is a constant danger, and together with the adhesions render the whole operation anxious and arduous.

Carcinoma of the sigmoid readily gives rise to obstruction, and this may occur suddenly without any marked antecedent symptoms. In such cases the sensitive distended cæcum may simulate the site of stricture. Should the rectum only contain a small enema the correct diagnosis may be reached, even when distension prevents palpation. The value of a rectal examination must be insisted upon in every case. The more closely the stricture approaches the ileum the less likelihood is there of obstruction, for the fluid and gaseous contents of the small intestine may freely pass through the narrow channels as in the preparation Fig. 2 Pl. III.

The prognosis seems to be good in cases where there has been early diagnosis and interference. The long periods for which the disease may remain strictly local is remarkable. The neoplasm tends to run at right angles to the axis of the canal, and not parallel to it as is frequently seen in the œsophagus and stomach. Secondary affections are most common in the liver. If the patient escape recurrence for twelve months the outlook is hopeful. The operative results then may be regarded as distinctly encouraging and are capable of greater improvement. Early exploration in cases of doubtful diagnosis is demanded. The dangers of an attack of obstruction cannot be exaggerated in view of the cardiac failure so often seen following auto-intoxication.

It remains to thank the various friends who have recognised the value of early surgical intervention, and Mr Miles, Dr Johnston, Dr Jeffrey and the House Surgeons for their valuable aid at many a tedious case.

TABLE OF CASES.

A.—*Ileo-Cæcal Carcinoma—Resection—Primary Suture.*

No.	Sex.	Age.	Date.	Site.	Result.	Present State.
1.	Miss K.	47	1893.	Ileo-cæcal.	Healed.	Well.
2.	Mrs P.	40	Feb. '97.	Asc. Colon.	Healed.	Died Mar. '98.
3.	Mr M.	67	Nov. '96.	Asc. Colon.	Healed.	Died Jan. '97.
4.	Mr C.	28	Aug. '96.	Appendix.	Healed.	Well.
5.	Mrs M.	40	Ap. 23, 1901.	Asc. Colon.	Died Apr. 25.	
6.	Mr A.	32	April 1901.	Ileo-cæcal.	Healed.	Well.

B.—*Resection—Artificial Anus—Incomplete Operation.*

No.	Sex.	Age.	Date.	Site.	Result.	Present State.
7.	Mrs C.	59	Dec. 1901.	Asc. Colon, &c.	Died.	

C.—*Lateral Anastomosis—Ileum to Ascending Colon.*

8.	Mr F.	35	May 1901.	Ileo-cæcal.	Healed.	Well.

D.—*Ileo-Cæcal Stricture, Non-malignant—Resection—Primary Suture.*

9.	Boy S.	9	1899.		Healed.	Well.

E.—*Resection—Secondary Suture.*

10.	Nellie S.	7	Resection, 1st June. Suture, 5th June '95.		Healed.	Well.

F.—*Carcinoma of Sigmoid Flexure—Resection—Primary Suture.*

11.	Mr G. S.	36	1896.		Healed.	Died Feb. '97.
12.	Mrs M'K.	58	6th Sept. 1896.		Died 5th Oct.	
13.	Mr S.	38	June 1900.		Healed.	Well.

G.—*Resection—Secondary Suture.*

14.	Mr D.	60	Colostomy, Mar. '95. Resection and Suture, April.		Healed.	Died 1896.
15.	Mrs T.	65	Colostomy, Mar. '95. Resection and Suture.		Died.	
16.	Mrs J.	47	Colostomy, 5th Sept. 1900. Resection and Suture, 8th Sept.		Died., 10th Sept.	
17.	Miss B.	39	Resection and Colostomy, 3rd Oct. 1900. Suture, 15th Oct.		Healed.	Well.
18.	Mr J.	55	Colostomy, Dec. 1898. Resection and Suture, Feb. 1901.		Healed.	Well.
19.	Mr B.	56	Resection, Colostomy, Jan. 30, 1901. Union, March.		Fæcal Fistula.	Died April.

H.—*Resection—Artificial Anus—Incomplete Operation.*

20.	Miss B.	35	Resection, Colostomy, July 30.		Died Aug. 14, 1901.	

2. CLINICAL AND EXPERIMENTAL OBSERVATIONS UPON GENERAL PARALYSIS

By LEWIS C. BRUCE, M.D., F.R.C.P.ED., Physician-Superintendent, Perth District Asylum, Murthly, N.B.

IN this paper I cannot enter into full details descriptive of the disease known as general paralysis of the insane. It is necessary, however, to define general paralysis as a disease

marked by progressive degeneration of all the bodily and mental functions, commencing first with those most highly developed and recently acquired, and ending with complete wreckage of mind and body. The course of the disease varies greatly according to individual resistance. Some patients show mental symptoms, becoming maniacal or melancholic, before motor symptoms become prominent, while others present motor symptoms sometimes for years before any mental peculiarities develop. The disease is regarded as incurable; but remissions, often of such completeness as to simulate recovery, are by no means uncommon. The typical disease is further peculiar in having several stages. The first stage, in which the patient is often actually ill, in which febrile temperature, loss of body weight, restlessness, excitement, and sleeplessness are the outstanding symptoms; the second stage, in which the patient becomes fat, facile, lazy, and liable to congestive seizures; the third stage, when the patient again loses weight, becomes mentally demented and physically paralysed.

The causes of general paralysis are very variously stated, but the opinion that syphilis, if not the only cause, is certainly one of the chief causes is at present gaining ground both in this country and abroad.

The following observations were made upon patients suffering from undoubted general paralysis. I would divide these observations into—

I. Those observations made upon patients in whom the disease was steadily progressive.
II. Those observations made upon patients in a state of remission.
III. Those concerned in an attempt to produce remission.

I. Patients in whom the Disease was Progressive.

The system which I believe to be invariably affected throughout the whole course of general paralysis is the alimentary system. I have never yet seen a case of early general paralysis in which one did not meet with intestinal and gastric disorders—obstinate constipation, biliousness, capricious appetite, gastric crises, failure of digestive and assimilative power are the commonest symptoms. In the second stage

bulæmia may be and generally is added to the symptoms of the first stage, while in the third and last stage constipation alternating with diarrhœa is the rule. These symptoms have been commented upon by innumerable writers, but their significance has not been appreciated, nor do I think any observer has ever realised that they run from beginning to end of the disease, as they are so frequently masked by mental symptoms. When you have heard from Dr Ford Robertson the pathology of the alimentary tract in general paralysis you will agree that the alimentary symptoms as a group are very important symptoms.

Frequent observations have been made upon the temperatures of cases of general paralysis. Savage believes that there is nothing characteristic about the temperature in general paralysis. Clouston believes the febrile attacks so common in the course of the disease are maniacal temperatures. Mickle, in an exhaustive treatise, draws many conclusions from the temperatures he observed, which for want of space I cannot repeat here. I have observed the temperatures of cases of general paralysis for several years, making continuous charts of four, five, and six months' duration. I admit there are some grounds for the diverse statements made regarding the temperatures in this disease. Some few cases, though steadily progressive, never have a temperature above 98·4° during the first and second stages of the disease; in others the temperature is an irregular one, febrile attacks occurring at long intervals. These peculiarities are explainable on the grounds that individual resistance to disease varies, and remissions in the course of the disease are very common. As the result of my observations I strongly endorse Macpherson's opinion that the most characteristic temperature in general paralysis is a recurrent febrile attack every one or two weeks. I have endeavoured to represent what I believe to be the characteristic temperature of the disease in the accompanying chart.

Upon admission there is probably mania with a febrile temperature of about a week's duration, then there is a remission for, say three weeks, and again a febrile attack, with or without mental symptoms, followed again by a period of remission, which may be months in duration, and take the patient well into the second stage. When the second stage is established, the febrile attacks become more frequent and the remissions

shorter, and this may continue for months. As the second stage gradually passes into the third stage, the temperature tends to become subnormal, the febrile attacks of the second stage being represented by a gradual rise from subnormal to normal, but 99° F. is not often reached, the explanation being that the patient is becoming exhausted, and does not react so vigorously to the disease as previously. When the third stage is fairly entered the temperature becomes very irregular—to-day slightly febrile, to-morrow subnormal, and so on, the outstanding feature being the irregularity. I have purposely omitted the febrile reactions to congestive seizures and apoplectiform attacks, as these only complicate the main issue, and must be regarded as accidents in the course of the disease. Regarding such a temperature chart as a whole, the conviction is forced upon one that such recurrent febrile attacks can only be regarded as due to some recurrent cause, and that cause is a toxic one. Each febrile attack represents the resistive reaction of the body to some toxic substance, and each inter-febrile period an intermission when the resistive powers of the patient have subdued the action of the toxin. Kanthack considers it a rule "that previous to recovery from bacterial products an increase in the number of leucocytes takes place." It is hardly necessary to say that many workers have examined the blood in general paralysis; but such observations were single ones, made upon different patients, and gave the most irregular results. I therefore counted the leucocytes and took the temperatures daily, or every second day, of some six cases of general paralysis for three or four months continuously, and a glance at the dotted line on the chart will show that to every rise of the thermometer there was a corresponding rise of leucocytes. In the first stage the hyperleucocytosis was often very high, sometimes 20,000 to 30,000 her c.mm., and the higher and longer continued the leucocytosis the longer was the remission that followed the attack. In the second and early third stage the leucocytes still rose with each rise of temperature, but the reaction was not so vigorous, and between the attacks the leucocytes tended to fall lower than in the first stage. The remissions were also shorter. In the later period of the third stage the leucocytosis resembled the temperature in being very irregular, but did not necessarily coincide with the irregu-

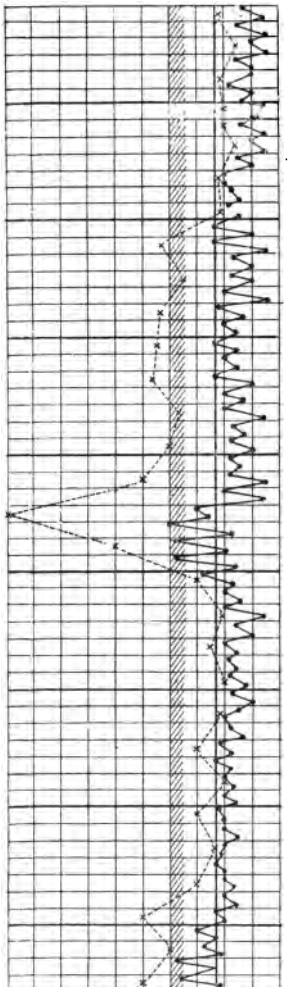

CHART 1.—First stage. Patient excited, restless, suffering from vivid hallucinations of sight and hearing, sleepless, physically thin, wasted, cachectic; bowels obstinately constipated; temperature shows two febrile attacks with hyperleucocytosis; the period of remission following febrile attack No. 2 was prolonged into the second stage.

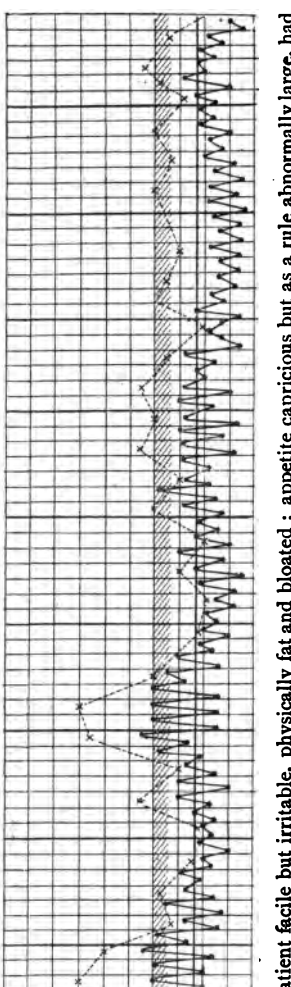

CHART 2.—Second stage. Patient facile but irritable, physically fat and bloated; appetite capricious but as a rule abnormally large, had several attacks of gastric crises; temperature shows a recurrent febrile attack every three or four weeks with a corresponding hyperleucocytosis; the periods of remission are short.

larities of temperature. There were practically no periods of remission. Those progressive cases of the disease in whom there was no recurrent febrile temperature nevertheless presented every now and then a hyperleucocytosis. These periods of hyperleucocytosis coincided with the periods in which there was advance in the disease, and following upon these periods of hyperleucocytosis there were remissions just as in those cases with distinct febrile symptoms. Each examination of the blood was made at the same hour every day and upon an empty stomach. During the period when these observations were being made, we admitted into Murthly Asylum an acute early case of general paralysis. I was examining a fresh specimen of this man's blood one morning, and noticed a motile bacillus. A film specimen stained with methylene blue showed what looked like streptococci. Film specimens were sent to Dr Ford Robertson, of the Scottish Asylums Pathological Laboratory, for examination, and he confirmed my observations. This patient also developed hæmaturia. One ounce of his urine obtained with antiseptic precautions was centrifugalised, and in the deposit so obtained we found the same motile organism I had at first noticed in the blood, but no streptococci. Before I could obtain nutrient media, the streptococci and bacilli disappeared from the blood and urine. This is the only case of general paralysis in which I have actually found living organisms in the blood. I regard it as an accidental occurrence, but it may be an accident which occurs much oftener than is supposed if the condition is looked for.

Dr Ford Robertson at this time drew my attention to the fact that the intestinal mucus in cases of general paralysis swarms with bacteria, chiefly the bacillus coli. The same fact was recorded by Cowan in 1895. As the motile bacillus observed in the blood and urine of the acute case of general paralysis above mentioned resembled the bacillus coli, I tested the blood serums of my general paralytic patients in various conditions of the disease, to see if they had the power of agglutinating bacillus coli. Dr Welsh, Pathologist to the Royal Infirmary, Edinburgh, kindly tested some of the serums for me, and supplied me with pure cultures of the bacillus coli, from which I made subcultures in nutrient broth. Dr M'Rae, Assistant Physician to the Royal Edinburgh Asylum, was also good enough to send me many speci-

mens of the blood of cases of general paralysis. The serums of ten progressive cases of the disease were tested as to their agglutinative power on bacillus coli in dilutions varying from 1 to 5 to 1 to 20, and no reaction was obtained.

II. Patients in a State of Remission.

I would divide remissions in the course of general paralysis into two classes:

1. *Remissions which follow upon Acute Attacks of Intercurrent Disease.*

I have seen remissions occur after acute erysipelas of the scalp, pneumonias, severe carbuncles, etc. Such remissions are preceded by a high and long continued hyperleucocytosis,

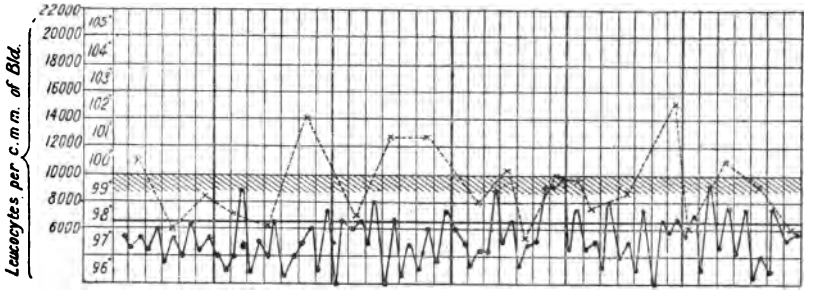

Chart 3.—Third stage. Patient demented and physically weak; frequent attacks of diarrhœa; temperature irregular; leucocytosis irregular; no periods of remission.

but I have unfortunately not been able to make further observations upon such cases.

2. *Remissions which occur Naturally and without Obvious Causes during the Course of the Disease.*

In such cases it is extremely hard to say when a patient, suffering from general paralysis, is in a state of remission, because, as I have pointed out, many cases, on account of individual resistive power, although they never show any marked progressive symptons, are still, if observed for months, steadily progressive. I therefore record only such cases as I have observed actually to improve mentally and physically in the course of the disease, and not those who merely remained stationary. I found in those cases that the powers of the alimentary system improved. The temperature was never febrile, but the outstanding feature was the fact that

the number of leucocytes in the early stage of the remission rose higher than they are in ordinary health, varying between 9000 to 12,000 per c.mm. of blood.

As the remission became fairly established the leucocytosis gradually fell to that of ordinary health, that is, between 6000 and 8000 per c.mm., when the subject is fasting. The blood serum also of such cases contains some antitoxic substance which is not present in patients in whom the disease is progressive, nor in the serum of healthy persons. The power of the blood serum to agglutinate B. coli in dilutions varying from 1 in 5 to 1 in 20 was tested in ten cases in states of remission; seven gave a definite agglutinative action. Nature then appears to check the disease and produce remissions by first producing a hyperleucocytosis, the leucocytes in their turn producing some substance of the nature of an antitoxin in the blood which protects the patient, for a limited time at least against further doses of toxin.

III. Attempts to Produce Remission.

My observations led me to believe that the majority of cases of general paralysis were due to toxic poisoning, the toxins being bacterial toxins absorbed through the stomach or intestines. I believed that each febrile attack represented the actual entrance of bacteria or the toxins of bacteria into the blood or tissues of the patients, and that during periods of remission the blood must possess some antitoxic substance. I tested the truth of these observations in the following way.

A case of general paralysis in the second stage of the disease, during a well-marked remission, had an attack of lumbago. I wet-cupped this patient with antiseptic precautions, and defibrinated the blood so obtained. I then placed two earlier and progressive cases of general paralysis in bed and injected subcutaneously into each of these patients 2 c.cm. of this defibrinated blood daily for three weeks. Patient No. 1 was in the early first stage. Patient No. 2 was in the early period of the second stage. This experimental treatment was conducted nearly two years ago.

Patient No. 1 became so far recovered that his friends insisted upon his discharge, and he accordingly left the asylum in February of this year. He called to see me last month. I

found that he still presented some motor symptoms of general paralysis, and mentally he was impaired. Still he has supported himself by his own efforts in his trade as a saddler since his discharge.

Case No. 2 has shown physical improvement but no mental change, and I think his brain is permanently damaged. The progress of the disease, however, in his case has been definitely arrested for nearly two years.

Four other cases of general paralysis, contemporaries of these two so treated patients, are now all dead, and it is a suggestive fact that these two men should have given the reaction they have done to treatment.

As it is obviously impossible to continue treatment on these lines, I believe, from the fact that the blood of patients in conditions of remission can agglutinate the bacillus coli, that small subcutaneous injections of blood serum of a horse immunised to B. coli, continued for three weeks or a month, may produce artificial remission in the disease, and I am giving this treatment a trial.

CONCLUSIONS.

My conclusions drawn from these observations and experiments are that :—

1. General paralysis is a disease directly due to poisoning by the toxins of bacteria whose point of attack is through the gastric and intestinal mucous membrane.

2. The poisoning is probably a mixed poisoning, but the bacillus coli is apparently one of the noxious organisms.

3. The result of treatment with serum taken from a case of general paralysis in a condition of remission and injected subcutaneously into an early progressive case, points strongly to the fact that some form of serum treatment is the proper treatment for this as yet incurable disease.

APPENDIX.

Observations made upon the Agglutinative Power of the Blood Serum of General Paralytics upon the Bacillus Coli.

Method of Investigation.—In every case a twenty-four hours' culture of the bacilli in broth media incubated at 98·5° F. was used. These cultures were made from a stroke culture on gelatine kept at ordinary room temperature. In every observation controls were used. The results were as follows:

1. The highest dilution in which, as a rule, agglutination occurred was 1 in 10, and even in this dilution agglutination was sometimes delayed for four or five hours. In one case only was the serum so potent as to agglutinate in a dilution of 1 in 100, but this was only a temporary condition.

2. Out of 18 control bloods, 3 (16·6 per cent.) produced a very slight degree of agglutination.

3. Agglutinative power was always absent from the serum of those cases in which the disease was progressive. The exception to this rule is that the blood of patients in congestive seizures agglutinates B. coli.

4. Agglutinative action was present in 70 per cent. of the serums of patients during periods of remission. When the remission was of long standing the serum was found in the same patient to vary in agglutinative power from day to day.

5. Agglutinative action was never a complete action. There always were between the clumps of bacilli motile bacilli apparently unaffected by the serum action.

6. A transitory agglutinative power was present in the serum of 60 per cent. of cases which were non-progressive, but which could not be said to be in a state of remission. This transitory agglutinative power was associated with conditions of hyperleucocytosis. The permanent agglutinative power of the serum of patients in a condition of remission was not necessarily associated with hyperleucocytosis, but is probably due to some antitoxic substance present in the blood. I do not regard this serum reaction as in any way diagnostic of the disease, but merely another link in the chain of evidence that general paralysis is caused by the toxins of intestinal bacteria, and that one of these noxious organisms is the B. coli communis.

3. OBSERVATIONS BEARING UPON THE QUESTION OF THE PATHOGENESIS OF GENERAL PARALYSIS OF THE INSANE

By W. FORD ROBERTSON, M.D., Pathologist to the Scottish Asylums

THEORIES OF THE PATHOGENESIS.

ALTHOUGH the various views that have been advanced in explanation of the pathogenesis of general paralysis of the insane are almost as numerous as the individuals who have

written upon the subject, those that can at present be regarded as occupying a place in the field of controversy permit, I think, of being grouped into two main opposing theories. There is now practically no dispute as to the fact of the toxic origin of the disease. Nearly all authorities recognise that antecedent syphilitic infection is the essential cause in a very large proportion of cases. Some, indeed, go so far as to maintain that without syphilis there can be no general paralysis, but there is still a large consensus of opinion in favour of the view that other toxic agents, more especially alcohol, lead salts, and highly nitrogenous foods, may, independently of syphilitic infection, determine the occurrence of the same morbid process. The great point of controversy is really the mode of action of the toxins. Owing to the long interval that commonly elapses between the date of the syphilitic infection and the onset of the cerebral disease, most authorities have felt a difficulty in admitting that there is a direct action of the syphilitic poison upon the brain. An endeavour has therefore been made to discover some indirect relationship between the original toxic agent and the morbid process that occurs in the nervous system. Out of this endeavour the two opposing theories to which I have referred have arisen. On the one hand it is asserted that the syphilitic poison produces upon the most highly developed nerve cells of the cerebral cortex a certain impression, in consequence of which these cells after some years become exhausted of their trophic energy, and thus undergo premature involution. On the other hand, it is contended that there is a toxæmia of general paralysis which is in some way brought about in certain individuals by antecedent syphilitic infection, and also, it would seem, by certain other pathogenetic factors of a different nature. According to some, this toxic action is exerted directly upon the cortical nerve cells; according to others, it induces in the vessels of the central nervous system certain structural alterations, in consequence of which the nutrition of the adjacent nervous elements is interfered with.

The theory of the premature involution of the cortical nerve cells is advocated especially by Mott and Krafft-Ebing. It is stated by Mott (1) in the following terms:

"In general paralysis there exists a primary progressive decay of the nerve cell which ontogenetically and phylogenetically may be looked upon as a regressive metamorphosis, a

degenerative process which starts in the highest and latest phylogenetically developed structures, *e.g.*, the centres of verbal and written speech; the molecular layer with the tangential system of fibres and the association systems of the frontal and parietal lobes. This premature decay of the highest controlling structures is progressive and cumulative, it causes phenomena of irritation, manifested by mental and physical symptoms, such as motor irritation, with rise of blood pressure, cerebral anæmia, and venous stasis, local and general auto-intoxication respectively by products of degeneration and imperfect metabolism. A vicious circle is established which continually is enlarging."

The theory of the existence of a chronic toxæmia is virtually a further elaboration of the view that the cerebral vascular lesions constitute the first anatomical alterations in general paralysis, although, as I have indicated, some authorities maintain it independently of this view. Most of the older observers who attached special importance to the early occurrence of vascular changes in the brain, merely regarded these changes as representing the initial stage of a chronic meningo-encephalitis. Although several previous writers had suggested the possible dependence of general paralysis upon some special forms of toxæmia, Angiolella (2) appears to have been the first to formulate this theory as a definite conclusion, based upon the results of original clinical and pathological observations. He has certainly been its most able advocate, and his name may, I think, without injustice to any other author, be specially associated with it. In three papers published in 1894-1895, he defended the vascular theory, and contended that the changes in the walls of the cerebral and other vessels could only be accounted for by the presence of toxic agents in the blood, which he believes were either products of syphilitic infection, or certain special poisons, such as alcohol, nicotine, etc. He further expressed the opinion that the toxic substances were either simply of a chemical nature, the result of the growth of infective agents, or products elaborated by the tissues. He left it to future investigators to determine their exact nature and source.

The Theory of Premature Involution.

It is scarcely necessary for me to mention the main arguments by which Mott defends the theory of premature involution,

for they are already well known. I have previously pointed out what appears to me to be certain strong objections to the acceptance of this theory, and defended the views of Angiolella on the ground of personal observation (3). The more recent observations which I wish to record in this paper have served only to deepen my conviction that general paralysis depends upon a chronic toxæmia, which produces its effects mainly through implication of the vessels of the central nervous system, and that the theory of the premature involution of the nerve-cells is untenable. I believe that the toxæmia is of a far more special nature than has hitherto been thought, and that it is essentially different from the primary toxic conditions by which it is brought about.

The following are what appear to me to be some of the chief difficulties in the way of accepting the theory of the failure of the specific vital energy of the cortical neurons. It does not adequately account for the changes that take place in the nervous system. The profound alterations that constantly affect the arterioles and capillaries cannot be satisfactorily explained as the result of the action of irritative products formed in consequence of degeneration of the adjacent nervous elements, for experimental secondary degeneration of the nerve cells and fibres is not attended by similar irritation of the neighbouring vessels, and in many cases of acute mania of some weeks' duration, there is a far more rapid destruction of cortical neurons than in general paralysis but no corresponding vascular changes. Nor does the theory account for the widespread implication of the nerve cells. As Angiolella and others have pointed out, the whole nervous system is involved in some measure, including even the sympathetic ganglia and cranial and spinal nerves. A premature involutive process ought to select certain functional groups of neurons, but motor, sensory, and associative elements are indiscriminately involved. The clinical and pathological picture of general paralysis has no true resemblance to normal senile involution. What the disease does in a measure simulate is that form of senile insanity which is clearly dependent upon a chronic toxæmia and structural alterations in the cerebral vessels. The theory does not explain the unquestionably close relationship between general paralysis and tabes dorsalis, for the fact is now clearly established that the latter disease does not depend

upon degeneration of the cells of the posterior root ganglia. It does not explain how it is that general paralysis may develop independently of previous syphilitic infection. It does not accord with the phenomena of successive remission and recrudescence of the more active phase of the disease. Lastly, the theory does not account for the remarkable association of general paralysis with various forms of bacterial infection.

On the other hand, the evidenee that has now been collected in support of the theory of the occurrence of a chronic toxæmia is, beyond all question, exceedingly strong. Moreover, two objections to this explanation of the pathogenesis of general paralysis, which at one time had certainly very great weight, can now, I think, be disposed of in a perfectly satisfactory manner. The objections to which I refer are, first, that general paralysis cannot be the result of a chronic toxæmia, because the operation of such a toxic agent would not account for the fact that the nervous system is primarily and specially affected; and, secondly, that such a conception of the pathogenesis of the disease is obviously inconsistent with the fact that certain of the most highly-developed and most recently-evolved functions of the brain are the first to become impaired —"*e.g.* the centres of verbal and written speech." It is to be noted that the ground of the first of these objections is not strictly in accord with fact, for it has now been very clearly demonstrated that the essential tissue changes in general paralysis are not confined to the nervous system; but, even if the objection is allowed its full weight, it can still, I maintain, be satisfactorily met. The explanation lies in the fact of the peculiar structure of the vessels of the central nervous system. The capillaries differ from those of other organs in having a special adventitial coat. They are thus placed in the position of the small arterioles, and rendered liable to the same morbid changes. Another fact that appears to have considerable importance in this connection is that the adventitial coat is composed of elements that are much more highly developed than the corresponding elements in other vessels. The fibrils are extremely elastic, and yet differ in certain respects from ordinary yellow elastic fibres. I hold that it is owing to these peculiar structural features that the vessels of the central nervous system tend to be specially affected by certain toxins.

The inconsistency of the theory of a chronic toxæmia with the early impairment of the highest cerebral functions is really only on the surface. The phenomenon is exactly such as we should expect from the action of toxins that specially injure the vessels of the nervous system. It is now clearly established that the amount of blood supplied to the different parts of the brain is proportionate to the amount of work they do; functional activity is accompanied by local hyperæmia, rest by comparative anæmia. Couple with this the now clearly established principle that the action of any toxin is directly proportionate to the quantity that is brought into contact with the tissues. The parts of the central nervous system that first give way under the action of the toxæmia of general paralysis are precisely those that have exhibited the greatest degree of functional activity, and have consequently received the largest amount of blood, and have had brought to them the greatest quantity of toxin. Hence their arterioles and capillaries are among the first to become altered in structure, and impairment of the nutrition and functional action of the neighbouring nervous elements is the result.

The Theory of Chronic Toxæmia.

It is impossible for me to enter fully here into the case that has already been made out in support of the theory of a chronic toxæmia. I can only very briefly indicate some of the more important observations that have been recorded. It is asserted, and it seems to me with perfect reason, that vascular lesions of the nature of those that constantly occur in general paralysis can only be produced by toxins circulating in the blood. Angiolella has insisted strongly upon this fact, and has made some important observations which go to confirm it. He has found that the vascular changes are not limited to any particular region of the nerve centres, but occur in some degree in every part of them, including the sympathetic ganglia; further, he has observed that such changes are not confined to the central nervous system, but take place also to an important extent in the hepatic arteries, portal veins, renal arteries, and nutrient vessels of the aorta. Numerous observers have found that general paralytics are specially subject to various forms of chronic nephritis, which at the present day we can only regard as a manifestation of toxic action. The

urine almost constantly displays various important abnormalities. In advanced cases the bones and cartilages always manifest certain gross structural changes which can only be inadequately accounted for by toxic action, The bones generally show a high degree of osteo-sclerosis ; in the ribs the alterations very commonly take the form of osteo-porosis. The cartilages constantly undergo well-marked degenerative changes which tend to be followed by vascularisation. D'Abundo (4) has found that the blood has an increased toxicity, whilst at the same time it exhibits other distinct changes.

Numerous observers, including A. W. Campbell (5), and Orr and Cowen (6) in this country, have found degenerative changes in the cranial and peripheral nerves, which certainly suggest the operation of a toxæmic process. In this connection I think special importance is to be attached to the observation of Orr and Cowen that the changes in the cranial nerves have the characters of a primary and not of a secondary degeneration. This fact implies that the change is brought about by the action of a toxin, and not by destruction of the nerve cells from which the fibres originate. There are also numerous observations that go to show that general paralysis has some special relationship to bacterial infection. D'Abundo and Piccinino (7) have found various forms of bacteria in the blood. The latter has also observed in the cerebral cortex a bacillus resembling that described by Lustgarten in syphilis. Grimaldi (8) examined the blood and urine in ten cases, and obtained a streptococcus from the blood of one, and various organisms, mostly bacilli of the colon group, in the urine of four. The latter observation, put side by side with the fact of the great frequency of gastro-intestinal disturbances in general paralysis, induced him to suggest that in some cases of the disease the intoxication is of gastro-intestinal origin. Probably the more remarkable researches of this nature are, however, those most recently recorded by Montesano and Montessori (9). These observers made an exhaustive bacteriological examination of the cerebro-spinal fluid, obtained by lumbar puncture, from eleven cases of general paralysis. In three cases they could not obtain a culture of any organism ; in eight they succeeded in isolating either one or two forms. In four of these cases they obtained a peculiar bacillus, to which they applied the

name of "bacillus viscosus"; in three they isolated streptococci, in two they found staphylococci and sarcinæ, and in one, the subject of severe epileptiform attacks, the bacillus of tetanus.

I now come to the new observations that I have to record.

The Source of the Toxins.

In searching for the source of the toxins I directed my attention first to the liver. The examination of this organ in a series of cases served, in the first place, to satisfy me of the accuracy of Angiolella's observation as to the frequency of irritative changes in the walls of the hepatic arteries and portal veins, as well as regarding the frequency of well-marked pigmentary and fatty changes in the liver cells. I also found that the lumen of the hepatic arteries was very frequently greatly narrowed by thickening of the intima, but the observations of Ainslie seemed to show that this condition was merely a part of a more or less general thickening of the intima of the arteries throughout the body. I felt obliged to conclude that there were no changes in the liver of such a special character as to render it probable that general paralysis depended upon some form of autointoxication of hepatic origin. The prominence of the irritative changes around the portal vessels and bile ducts seemed to me to indicate that some irritant was being absorbed from the alimentary tract. I accordingly undertook an investigation of the condition of the stomach and intestines of general paralytics. Before recording the results of this investigation, I would refer to some previous observations that have been made upon the condition of the digestive organs in this disease.

Dr Bruce has already described the typical signs of gastro-intestinal disturbance that may be observed, and has referred to the fact that most writers on general paralysis make mention of the common occurrence of various disorders of the alimentary tract, to which, however, they attach only a secondary importance. Mickle (10) specially remarks upon the occurrence in the later stages of general paralysis of troublesome, and at times intractable, diarrhœa, associated with local "muco-enteritis, mucous congestion, or ileo-colonic ulceration." Agostini (11) states that at the beginning of the disease there is very frequently want of appetite, eructations, and constipation;

in the second period, accompanying mental exaltation, there is generally voracious appetite and increased constipation; in the final stages of the disease, he says, the gastro-intestinal disturbances are increased, and may lead to colliquative diarrhœa. Numerous observers have recorded cases in which general paralysis was complicated by either gastric or intestinal hæmorrhage.

Dr T. P. Cowen (12) has recently given a very full description of the "atrophic intestinal affections of the insane, based upon the post-mortem examination of the intestines in thirty-two cases, mostly of general paralysis, in which diarrhœa had been a prominent clinical feature. He excluded from his investigation all cases in which there was the least suspicion of tubercle, enteric fever, or dysentery. He found changes of a very distinct character, including specially such conditions as follicular ulceration of the ileum, catarrhal enteritis, ulcerative colitis, and congestion. He concluded that these lesions are of a "dystrophic or tropho-neurotic origin." Galante (13) has studied the chemical characters of the gastric contents in a number of general paralytics. He found that there constantly existed evidence of the occurrence in a severe form of that condition termed by Hayem "hyperpeptic parenchymatous gastritis." There was constantly a condition of hyperpepsia, frequently accompanied by hyperchlorhydria. He remarks upon the absence of those severe subjective and functional disturbances which commonly accompany a similar form of disorder of the gastric digestion in other persons. He found that there was evidence of the occurrence of abnormal fermentations which indicated the production of toxic substances analogous to ptomaines. When the patients were at their best, the amount of free hydrochloric acid was high; when they were in a torpid and stuporose state, it was less. When there was great excitement, accompanied by a furred condition of the tongue, sordes upon the teeth, fœtid breath, and anorexia, the amount of free hydrochloric acid became subnormal, falling even to zero, whilst at the same time the organic chlorides were increased. It is a noteworthy fact that in several instances he was obliged to cease the examination of the gastric contents, owing to the circumstance that the moment he introduced the stomach tube there was very profuse bleeding, which he regarded as at least an indication of

hyperæmia and easy lacerability of the gastric mucous membrane. These observations are all the more valuable on account of the fact that Galante accepted it as a postulate, the accuracy of which he never seems to have doubted, that the functional disturbances of which he found so much evidence were the result of the morbid processes occurring in the central nervous system.

THE GASTRO-INTESTINAL TRACT IN GENERAL PARALYSIS.

My own systematic investigations into the condition of the gastro-intestinal tract in general paralysis extend as yet to only twelve cases of the disease. In certain respects, however, the evidence of these cases has been so uniform that, considered in conjunction with the large amount of confirmatory evidence that has now been obtained, it justifies, in my judgment, the formulation of certain definite conclusions. The organs were examined after hardening in 5 per cent. formalin, which was introduced into the interior of the gut in order to fix the mucous membrane. Seventeen control cases were examined in the same way. In every case of general paralysis I have found chronic morbid changes of a very pronounced character. In eight of the cases there was distinct thickening of the wall of the stomach, and in two others there was a very marked degree of dilatation. In five cases the thickening of the wall of the stomach was associated with some thickening of the duodenum and upper portion of the jejunum. In three cases in which there was phthisis there was extensive tuberculous ulceration of the ileum. In the remaining nine cases there were various other lesions of the small intestine. In four cases, near the lower end of the ileum, and affecting from a few inches to about two feet of its extent, there was a condition of thickening and induration of the gut, accompanied by very marked contraction of the lumen. The surface of the affected portion was for the most part covered with a thick, tenacious white or grey secretion. When this substance was removed an eroded condition of the mucous membrane was frequently revealed. In another case there were several areas of a similar character in a higher part of the ileum.

In the majority of the cases there was well-marked localised general congestion of the small intestine, accompanied by abundant secretion of mucus. In five of these cases, in which

there was no tuberculous disease, the greater portion of the small intestine, when held up to the light, presented a peculiar spotted appearance, which did not occur in any of the control cases examined. The spots were of a dark-brown colour, and about the size of the head of a common pin. They were accompanied by a slighter degree of irregular, diffuse brown staining. The mucous membrane of the duodenum and jejunum in one case, and that of the jejunum alone in another, presented an appearance which suggested that the surface had been powdered over with fine white sand. In most of the cases the valvulæ conniventes were entirely absent from the lower half or so of the ileum, and the mucous membrane had a smooth, atrophic appearance. In one case the valvulæ conniventes of the greater portion of the ileum were represented only by a few fibroid ridges. The lymphatic glands in the neighbourhood of the stomach and duodenum were in some instances found to be considerably enlarged. In most of the cases the colon showed signs of chronic distension. It was generally dilated and sacculated, and its walls somewhat indurated.

On histological examination the stomach presented evidence of long-standing catarrh, except in one case in which the preparations examined revealed no distinct abnormality. In this case the intestinal alterations were very pronounced. The catarrhal changes consisted chiefly in thickening of the mucous membrane, proliferation of the gland cells, especially of those near the inner surface, more or less dense infiltration of the mucous membrane with leucocytes, great increase in the number of fixed connective tissue corpuscles, increase in the amount of interglandular fibrous tissue, and coating of the surface with a layer of mucus containing very large numbers of leucocytes and disintegrated epithelial cells. There was generally evidence of very active secretion of mucus. In some areas the normal arrangement of the glands was entirely lost in consequence of the proliferative changes. In several cases small erosions of the mucous membrane were observed, extending often to very near the submucous layer. Around these erosions there were very acute reactive changes. The mucus on the surface always contained abundant bacteria. Amongst the proliferated and degenerated cells of the ducts and glands, bacteria of various kinds were demonstrable in

several instances. In one case in which death had occurred from septicæmia of sudden onset and rapid course, a short bacillus was found in great numbers in the wall of the stomach, and in the blood. The thickening of the duodenum was found to be associated with catarrhal changes similar to those in the stomach. The tuberculous lesions observed in the ileum do not require description, but it is to be remarked that in each instance they presented evidence of having run a very slow and chronic course. The non-tuberculous thickening of the wall of the lower part of the ileum was found to be due to chronic catarrhal changes in the mucous membrane, accompanied by fibroid overgrowth in the other coats. The mucous coat was thrown into irregular folds, the crypts of which were filled with mucus, cellular *débris*, and bacteria. The villi had disappeared and almost every remaining gland cell was seen to have been actively secreting mucus. There was a very pronounced degree of cellular infiltration of the mucous membrane and increase in the amount of fibrous tissue. The appearance of brown mottling was found to be due to greatly dilated vessels, chiefly in the submucosa, associated frequently with enlargement of the solitary lymph follicles. The appearance resembling fine white sand on the surface of the duodenum and jejunum was found to depend upon an atrophic and fibroid condition of the villi in an intestine the surface of which had not undergone post-mortem change. Chronic catarrhal changes were seen in most sections of various parts of the jejunum and ileum. In the lower half of the ileum there were almost constantly distinct atrophic changes in the mucous membrane, which was commonly reduced to about half its normal breadth, whilst there was a paucity of villi. I have not yet studied the microscopic appearances in the large intestine.

Author's Conclusions.

Regarding the significance of these gastro-intestinal lesions, I would say at once that I maintain that they are not the cause of general paralysis of the insane. They are effects of the same toxic condition as that which induces the other lesions already enumerated. Probably not one of them is special to general paralysis. Nevertheless, I think that the fact of their constant existence in the well-marked form that

I have described helps us very materially towards a true conception of its pathogenesis. It seems to me that the view that the disorders of the digestive functions depend upon the cerebral condition is quite untenable. Histological examination shows most conclusively that the chronic catarrhal changes are a reaction to irritants that bathe the internal surface. When we take into consideration all the known facts bearing upon the question, I think we may logically infer that the toxæmia of general paralysis is of gastro-intestinal and bacterial origin. The clinical and anatomical evidence of gastro-intestinal irritation, the irritative changes in the portal spaces, and the fact that so far it has been found impossible to localise the origin of the toxins in any other part of the body, all point to the gastro-intestinal tract as the place in which the toxins are formed. The disturbances of temperature that constitute a part of the clinical picture of the disease, the fact of their constant association with the profuse leucocytosis observed by Dr Bruce, the frequency with which bacteria of various kinds may be isolated from the blood, cerebro-spinal fluid, and urine of the general paralytic, and the fact that in the great majority of instances these patients finally succumb to a general invasion of the tissues by bacteria, are indicative of a struggle between the forces upon which natural and specific immunity depend, on the one hand, and some bacterial foes on the other.

I conclude with Dr Bruce that general paralysis depends upon a condition of gastro-intestinal autointoxication which results from excessive growth of the micro-organisms which normally inhabit the alimentary tract. I think there are grounds upon which we may definitely exclude the possibility that the disease depends upon the action of any single form of micro-organism. At the same time there is reason to believe that certain forms obtain an ascendency, especially some belonging to the "colon group." There is evidently a partial breakdown of those forces by which any harmful development of the intestinal organisms is normally prevented. I have looked into the question, and, as far as I can ascertain, no one has ever given a satisfactory explanation of how it is that we inhibit the growth of bacteria in the alimentary tract. Some writers attribute this inhibitory action merely to the hydrochloric acid of the gastric juice and to the bile. Another view

that I have found stated is that an excessive growth of bacteria is prevented by the accumulation of the toxins they produce. It seems to me that these explanations entirely miss the essential fact. Inhibition of the growth of the bacteria that are constantly present in the alimentary tract can only be satisfactorily explained as the result of the same forces as those by which the growth of bacteria is prevented in the living tissues, namely, by the action of leucocytes, alexins, and antitoxins. As important local auxiliary forces, chiefly mechanical in their action, there are the surface epithelial cells and the mucous secretion. Alexins and, especially under certain circumstances, antitoxins are contained in the secretions of the whole alimentary tract, beginning with the saliva. The food is mixed with these secretions, and in normal conditions the quantity of inhibitory substances contained in them is sufficient to prevent any harmful development of bacteria. Now there is conclusive evidence that in the general paralytic, even at an early stage of his disease, there is a weakening of the natural resistance to bacteria, or of the natural immunity. Such weakened power of resistance will manifest itself especially in the alimentary tract, in which the inhibitory substances are in a state of comparative dilution. Hence there will be an increased growth of bacteria and an excessive formation of their toxins. There will be set up a catarrhal state of the gastro-intestinal tract, which is really the expression of local effort on the part of the human organism to hold the bacteria in check. Any sudden excessive formation and absorption of toxins will cause a rise of temperature, general and local leucocytosis, a pouring out of leucocytes and alexins into the alimentary tract, a formation of specific antitoxins, and consequent arrest of the further growth of the bacteria. The specific antitoxins will for a time specially serve to hold under control those forms that chiefly threatened their host. When the specific antitoxins become exhausted these same bacterial forms may again become aggressive, and the whole process is repeated. Corresponding to these alternate waves of toxic action and tissue reaction, there are the clinical phenomena of alternate exacerbation and remission of the more active manifestations of the disease. Probably in most cases the bacteria are able to invade the proliferated and

degenerated epithelial cells at certain places, more especially, it would appear, the stomach and lower part of the ileum. The associated thickening of the tissues is exactly comparable with that which occurs in the walls of the bladder in certain forms of septic cystitis. Indeed, this condition of the bladder is not uncommon in general paralytics.

When the toxins are absorbed in quantities too great to be destroyed or rapidly excreted, they affect the nutrition of certain of the tissues. The vessels of the central nervous system are specially sensitive to these bacterial toxins, and, for the reason already explained, the capillaries suffer in the same manner as the small arteries.

The toxæmia is probably often in some measure established for months or even years before the first symptoms of nervous disease manifest themselves. In some individuals the cerebral vessels give way earliest, and general paralysis is the result; in others the vessels of the cord are the more vulnerable; those that receive the largest quantity of blood—and therefore of toxin—suffer first and most severely, and the clinical and pathological picture of tabes dorsalis develops (14). If this view of the pathogenesis of these two types of disease should be confirmed by future investigations, it will almost certainly be found that there are other diseases which depend upon essentially the same toxæmia. In stating this conviction I am not really weakening the case in support of the special character of the toxæmia in general paralysis and tabes dorsalis, but merely recognising more fully than is generally done the great importance of individual differences of tissue reactivity in determining the localisation and intensity of the effects of various toxic conditions. It is now very clearly recognised that the general paralytic and tabetic usually come of a stock that has been prone to suffer from chronic vascular disease and its effects, such as apoplexy, atrophic cerebral softenings, senile insanity, aortic disease, aneurysm, etc. This implies that the mesoblastic tissues of which their vessels are composed proliferate and degenerate under the action of certain toxins more readily than those of other persons.

The part that syphilis plays in the pathogenesis of general paralysis and tabes is probably chiefly that of profoundly altering the natural immunity. The alteration is of some

kind that can be brought about by certain other toxic agents. In what this modification of the natural immunity precisely consists must, in the present position of knowledge, be largely a matter of conjecture. I wish, however, to advance in explanation of it a hypothesis which, although many unanswerable objections can easily be urged against it, serves to explain some of the most contradictory facts regarding these two diseases. This hypothesis is that the alteration in the natural immunity and the diminished general resistance to bacteria depends upon a partial exhaustion of the leucoblastic function of the bone marrow. It is now clearly established, through the researches of Kurloff, Ehrlich, Muir, and Rubinstein, that the polymorphonuclear leucocytosis are essentially derived from the bone marrow. Leucocytosis and general resistance to bacteria are, therefore, functions of this tissue. It seems improbable that leucocytosis can go on indefinitely. If this function of the bone marrow has excessive demands made upon it over a prolonged period, it must necessarily in time exhibit signs of insufficiency, and the result of such insufficiency will be a diminished general power of resistance to bacteria, which will manifest itself early in the alimentary tract. I believe that it has been shown that the second stage of syphilis is accompanied by a high degree of leucocytosis. Most of the other conditions that are known to favour the development of general paralysis and tabes are likewise causes of prolonged leucocytosis.

This hypothesis would explain the essentially progressive character of general paralysis. It might also account for the remarkable fact that it is the rule that the persons who develop general paralysis after syphilis have almost invariably suffered from the latter disease in an extremely mild form. This freedom from the severer phenomena of secondary syphilis may have been purchased at the cost of a long sustained and very active leucocystosis, whereby the organism of syphilis was kept well under control. This hypothesis might also explain how it is that general paralysis is unknown in countries in which syphilis is almost universal, as, for example, in Abyssinia. The explanation would be that a considerable degree of tolerance of the syphilitic poison has been acquired by the inhabitants of these countries, and that consequently the disease is not accompanied by an exhaustive leucocytosis.

I have examined the bone marrow in a number of cases of general paralysis, and have always found it much altered in structure. I am as yet, however, unable to express any opinion as to whether the changes present are of such a nature as to give support to this hypothesis of leucoblastic insufficiency.

Summary of Conclusions.

I would briefly summarise my conclusions as follows: (1) General paralysis is dependent upon the occurrence of a chronic toxæmia of gastro-intestinal origin; (2) the toxins are mainly bacterial and are formed in consequence of a partial breakdown of those forces by which the harmful development of the micro-organisms that constitute the ordinary flora of the alimentary tract is normally prevented; (3) the toxins are absorbed and tend specially to produce proliferative and degenerative changes in the vessels of the central nervous system; (4) these vascular changes tend to set in earliest in those parts of the brain that are relatively best supplied with blood, because their walls are brought in contact with the largest quantity of toxins; (5) tabes dorsalis is dependent upon the same form of toxæmia; (6) the part played by syphilis in the pathogenesis of general paralysis and tabes dorsalis is essentially that of altering the natural immunity; (7) there is some evidence in favour of the hypothesis that this alteration in the natural immunity is dependent upon commencing exhaustion of the leucoblastic function of the bone marrow; (8) the treatment of general paralysis and tabes dorsalis should be directed primarily to the correction of the disorder of the alimentary tract; (9) probably the only means by which it will be found possible to check the excessive growth of the gastro-intestinal bacteria is that of the employment of specific antitoxins; (10) to arrest the disease by such means may be more practicable than would at first sight appear, because it is probable that the specially injurious toxins are the products of only a few bacterial forms.

[I have especially to thank Dr Clouston and Dr J. Douglas Macrae for the facilities they have given me in obtaining tissues from cases examined post-mortem at Morningside

Asylum, and also Drs Kerr and Primrose, Assistant Physicians at Hartwood Asylum, for material from two cases.]

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Meeting X.—July 3rd, 1901

MR A. GORDON MILLER, *President, in the Chair*

I. ELECTION OF MEMBER

H. de M. Alexander, M.D., Perth District Asylum, Murthly, was elected an Ordinary Member of the Society

II. ORIGINAL COMMUNICATION AND DISCUSSION

SOME NOTES ON ALCOHOL IN ITS MEDICAL AND SCIENTIFIC ASPECTS

By G. SIMS WOODHEAD, M.A., M.D., Professor of Pathology in the University of Cambridge

WHEN I was asked by your President to take part in this discussion, I gladly assented—(1) because, as many of you know, I take considerable interest in the question of Alcoholism; (2) because I know that your President is equally interested; and (3) because I expect to profit from the valuable contributions to the question to be made to-night, so valuable indeed that I may look upon my task as a comparatively light one.

Knowing, as many of you do, that I take what some may consider to be an extreme attitude in this question, and realising

that a man is much more easily made to appear convicted of error out of his own mouth when tackled by abler men than himself—although he may be thoroughly in the right—I shall, in dealing with the pathological conditions produced by alcohol, ask you to take much of what I have to say as put in the form of suggestion and question, although I may put my statements forward in somewhat dogmatic form. Many of you have looked at the question from quite other standpoints than that from which it has been my lot to survey it; and although we all who take part in this discussion may be able to answer most of the questions raised to-night to our own satisfaction, the cases of "conviction" may be somewhat isolated. Nevertheless, the question is one that may be ventilated with great advantage, especially as it is one concerning which such wide differences of opinion exist. It is always well to have clear issues, and before we can discuss this matter reasonably we must arrive at some agreement as to the category under which alcohol must come. Is it a poison? Is it a drug? Is it a food? Or, if it is all three, as some maintain, in which category does it play the most important rôle? As a food, or even as a drug, does it play a part anything like so prominent as it does as a poison?

We shall all agree, whether we take it or not, that it is a narcotic poison, and certainly that, as a pathogenetic agent, it is a protoplasmic poison. To this side of the question we must return. Is alcohol a drug? Certainly it is, but it is not a panacea for every ill to which flesh is heir, rivalling and even outstripping in the universality of its application the many "specifics" of the great army of quacks. If this be accepted, let us keep alcohol in its proper position as a drug, and let us use it and prescribe it as we should use any other medicament, not loosely, but with a definite end in view, and at any rate with some show of scientific precision. This, however, is beyond the scope of what I have to say, though I hope that the question will be raised by some of those who take part in this discussion.

The third question, Is alcohol a food? interests us greatly, because it appears to me that it is when alcohol comes to be looked upon as a food, and not as a potent drug, or a somewhat dangerous beverage, that it becomes responsible for the greater part of the diseases that are now ascribed to its action. Few physiologists nowadays deny that alcohol is oxidised in the body, and to that extent may supply a certain amount of heat, but that any sane man would put forward alcohol as able to supply the number of calories required by the body, and easily given by carbohydrates and fats, is now scarcely conceivable. The calculation has been made that such an amount of alcohol would be a constantly recurring certainly fatal dose.

Quite recently Dr John Madden,[1] criticising Professor

[1] *Am. Med.*, Phil., 1891, vol. i. pp. 209 and 258.

Atwater's experiments,[1] and the resultant teaching on the food value of alcohol, points out that alcohol differs from fats and carbohydrates, in so far that it is a violent protoplasmic poison, whilst they exert absolutely no toxic effect. In another direction, Ehrlich's[2] teaching on nutrition has a most important bearing on this question. The nutrition of cell protoplasm appears to result from a combination of certain proteid elements with the cells; for this combination certain bonds or links, or side chains as he calls them, must be present; in their absence there can be no application of these nutritive substances to the use and nutrition of the cell. All these links appear to be connected with the nitrogenous element, and consequently, unless this nitrogenous element be present, the nutritive combination cannot be effected. Alcohol does not contain this nitrogenous element. It certainly enters into combination with the protoplasm, but apparently in quite a different fashion from the nutrient substances ordinarily utilised by the cell, acting as a purely toxic agent and not as a nutrient substance. It interferes with the ordinary processes of metabolism, and to that extent is deleterious.

It is sometimes said that alcohol, like the fats and carbohydrates, even though it does not act directly as a nutrient material, may act as a respiratory food, and that it may, like these substances, be converted into a reserve supply of fat, upon which the organism may call whenever extra expenditure of energy is required.

Madden points out that there is absolutely no evidence of this, but that there is evidence, as pointed out by Weiske and Flechsig,[3] and Miura,[4] and Schmidt,[5] that, in place of protecting the nitrogenous tissues from undue waste, as do the carbohydrates and fats, alcohol actually hastens their waste, appearing to do this through its action as a protoplasmic poison, this being marked, however, only in patients or subjects who are not accustomed to the use of alcohol even in small doses.

One most important feature in connection with alcohol as a nutrient material and as a producer of disease, is the extraordinary avidity that it, like phosphorus, appears to have for oxygen; this it is that probably renders these substances prime factors in the determination of certain forms of fatty degeneration; it may also explain the wasting that takes place in the nitrogenous tissues of certain patients in whom there appears to be an abundant store of fat accumulated in the subcutaneous and other connective tissues. Under ordinary circumstances,

[1] *Harper's Mag.*, No. 605, p. 684.
[2] *Proc. Roy. Soc. London*, 1900, vol. lxvi. p. 424.
[3] *Jahresb. ü. d. Leistung. d. ges. Med.*, 1889, Bd. ii. S. 486.
[4] Quoted by Madden, *loc. cit.*
[5] "Inaug. Diss.," Greifswald, 1898.

the fat and carbohydrates taken into the body, or used up after being taken into the body, are readily oxidised by the respiratory oxygen, *i.e.* by the oxygen brought from the lungs by the hæmoglobin of the red blood corpuscles; they use up, in fact, the oxygen that is most readily obtained, and when the supply is exhausted then oxidation apparently no longer goes on. In the case of alcohol, however, we have a much more active process; it becomes oxidised, under conditions in which oxidation of fat and the carbohydrates could not take place; and, like phosphorus, in virtue of this property, it appears to divert to its own use oxygen, which, under normal conditions, would go to protect the nitrogenous tissues from undue waste.

In Madden's words, it "makes the demand for oxygen so great that it cannot be supplied by the normal process of respiration. Oxygen to supply this demand is therefore abstracted from the tissues, causing a derangement in their functions and metabolism." It interferes with the normal oxidation of carbohydrates and fat, and to that extent sets up abnormal metabolic processes, which end in the destruction of protoplasm and in the accumulation of the resulting fatty degenerative products in the protoplasm. Such fatty degeneration is perhaps seen best in the heart muscle, but what takes place in the heart may also take place in the muscular wall of the blood vessels of the intestine and of the other muscles and organs of the body. In all these cases, owing to the impairment of the nutrition of the protoplasm, and to the imperfect oxidation that takes place therein, the secondary fibrous and calcareous changes, so frequently associated with premature senility, are, I believe, directly due to this peculiar action of alcohol. The very readiness with which alcohol becomes oxidised, and with which the large amount of latent energy which it contains is set free in the body, renders its use dangerous, as these are the very features that characterise many of the products of the micro-organisms that induce disease. In this respect they all resemble phosphorus, and all of them offer us evidence of the danger of assuming that, because it is readily oxidised in the tissues, it is to be looked upon as a substance useful in the animal economy.

Ehrlich has pointed out that substances that conform much more closely to ordinary nutritive substances than do alcohol and phosphorus—the proteid toxines—are capable of combining with and acting as nutrient substances for protoplasmic cells, and that this renders their use dangerous. They combine with the cells as do ordinary nutrieut substances, but they have a secondary action, which interferes enormously with the normal metabolic processes of the cell and the degeneration of the cell results. In the case of alcohol we appear to have this degener-

ative action without any accompanying nutrient action or nutrient combination.

It is because of this want of the nutrient element that the protoplasmic cells do not in time become accustomed to the poisonous action of alcohol as they may to the poisonous action of the proteid toxines. As a result of tissue reaction between the toxine and the cell, the tissues of the body, as a whole, may in time become capable of withstanding enormous doses, thousands of times the original lethal dose of certain proteid toxines; but in the case of alcohol the process of immunisation, however careful and prolonged be the treatment, is only slightly —and then only apparently—increased; the degenerations, occurring as the result of the prolonged use of alcohol, affording marked evidence that the tissues at any rate do not become immune to its action in any very high degree.

It is important to bear in mind that alcohol acts as a cumulative poison, not only as to alcohol itself, but also in connection with the waste products of the body, and with certain other poisons of which arsenic, antimony, and phosphorus may be taken as types. There is still no great weight of evidence as to the experimental production of combined arsenical and alcohol poisoning; but, from the facts that have been adduced in connection with the recent epidemic of poisoning by arsenicated beer, it is evident that peripheral neuritis, resulting from the action of the combined poisons, is much more marked than when these poisons are allowed to act separately; there is, in fact, a cumulative action, the tissues "weakened" by one poison being more readily affected by the other. We shall probably have further light thrown on this subject when the full account of the recent epidemic appears, but it is interesting to note that what has been suggested in connection with this set of poisons —arsenic and alcohol—has been proved in connection with others, in connection with which experiments could be directly made.

For some time past it has been recognised by those who were observing the action of alcohol most closely, that it was in some way acting as a predisposing cause to specific infective diseases. M'Leod and Milles of Shanghai, in their admirable study of cholera,[1] insist most strenuously upon the importance of this factor in rendering patients susceptible to this disease. Later, in 1896, Abbott[2] pointed out that the normal resistance of rabbits to infection by *Streptococcus pyogenes erysipelatis* is markedly (and by *Bacillus coli communis* in a lesser degree) diminished by the use of alcohol, when given daily, to the stage of accute intoxication, and added that "the results of inoculation of alcoholised rabbits with the erysipelas coccus correspond in a

[1] *Rep. Lab. Roy. Coll. Phys. Edinburgh*, 1889, vol. i. p. 161.
[2] *Journ. Exper. Med.*, N.Y., 1896, vol. i. p. 458.

way with clinical observations on human beings addicted to the excessive use of alcohol when infected by this organism."

Not only did alcoholised animals show the effects of inoculation considerably earlier than did the non-alcoholised rabbits, "but, in the case of the streptococcus inoculations, the miliary abscesses produced were much more pronounced than were those that usually follow the inoculation of this organism." He adds that the diminished resistance here noted may be the result of structural alterations, consequent upon the direct action of the streptococcus on the tissues, as such alterations have now frequently been noticed.

It may be, however, that "the conspicuous influence of alcohol on the gastric mucous membrane" is the explanation of the marked loss in body weight, and that "the susceptibility induced by alcohol to excess is somewhat analogous to that induced by starvation, in which there is now ample proof that the resistance of animals to particular forms of infection is very markedly diminished."

These animal experiments were of considerable interest, and it was evident that they must be repeated by other observers, and under similar and different conditions. The following year, Delearde, in Professor Calmette's laboratory in Lille,[1] carried on a series of experiments to determine whether in animals poisoned by alcohol the different virus and toxines can, as under normal conditions, confer immunity. He found that animals treated with considerable doses of alcohol were less resistant to the action of the anthrax bacillus, tetanus toxine, and rabies poison; and also that it is a more difficult matter to render and maintain such animals immune to the action of these poisons than it is in the case of non-alcoholised animals. Not only is it the case during the time that the alcohol is being given, but in many instances the effects of the drug appear to continue for some time.

It is interesting to observe that Dr Delearde's attention was first directed to this matter, by the observation that an alcoholic patient, bitten by a rabid dog, appeared to be much more susceptible to the action of the rabies poison than another patient bitten and inoculated under otherwise much less favourable conditions.

All these experiments, however, had been carried on with animals treated with large and acutely toxic doses of alcohol, and were open to the objection that none but advanced alcoholics and actual drunkards are open to the susceptibility set up and maintained by the exhibition of large doses of alcohol.

Some two years ago, when visiting Professor C. Fränkel in the Pathological Laboratory at Halle, I was introduced to Dr

[1] *Ann. de l'Inst. Pasteur*, Paris, 1897, tome xi. p. 837.

Laitinen of Helsingfors, who, Fränkel said, was working at a subject in which he knew I was interested. These experiments have since been published,[1] and are well worthy of most careful perusal by those who wish to study this subject.

He took as the basis for his "small dose" experiments the amount of alcohol given as a nutrient substance or as a medicine, or both, in a sanatorium at Davos, *i.e.*, 40 to 60 c.c. of pure alcohol, or 0·5 to 0·8 c.c. per kilo., taking care to keep well within these quantities, and to use the alcohol in sufficiently diluted form. Sometimes the alcohol was given over periods of weeks or months before the inoculation or infection of the animal was commenced, and was continued after the infection; sometimes doses large enough to produce acute poisoning were given.

He used for his infecting material—(1) anthrax bacilli, Pasteur's "first vaccine," for susceptible animals (rabbits), whilst for the more resistant animals (dogs, fowls, and pigeons), a highly virulent culture was used; (2) tubercle bacilli in a glycerin emulsion, diluted with normal saline solution to 1 : 1000 and 1 : 10,000, 10 c.c., of one of these dilutions being injected into a vein; and (3) diphtheria toxine, of which the lethal dose can be easily measured. His first conclusion, as the result of nearly forty sets of most careful experiments, is that alcohol, however given, induces in the animal body a markedly increased susceptibility to artificial infection or poisoning, by the above infective and toxic agents; and he maintains that his experiments indicate that the use of alcohol in the treatment of anthrax, tuberculosis, and diptheria, at any rate, is not only useless but is probably actually deleterious. From a number of other experiments carried out with the greatest care, he comes to the same conclusion as do Abbott, Welch, and others—that the predisposing action of alcohol must be looked for in the production of abnormal conditions, as pathological changes in the various organs—alimentary canal, liver, kidneys, heart, nervous system, etc.

On the blood itself, alcohol appears to exert some direct effect, its alkalinity being slightly diminished and some leucopenia induced, during which latter there appears to be a slight decrease in the bactericidal power of the blood, and an increased susceptibility of the tissues to the action of infective agents.

Going beyond the general question of infection, and simply to bring forward evidence of the deleterious action of alcohol on the nutrition of the tissues, he points out that his experiments prove that pregnant animals and their offspring are markedly affected by the continued use of small doses of alcohol. The normal temperature and that in disease are, especially when large doses of alcohol are used, temporarily

[1] *Acta Soc. Sc. Fennicæ*, Helsingfors, 1900, vol. xxix. No. 7.

but often only very slightly lowered; but it appears that it is during this temporary lowering of the body temperature that we have the most favourable condition for the invasion of infective agents. As Pembrey[1] has pointed out, the fall in temperature is due to increased vascularity of the skin and increased activity of the sweat glands, the normal reaction to cold being paralysed by large doses of alcohol. Although he agrees with Laitinen, that alcohol taken in small quantities causes only slight depression—less than half a degree—the specific action is seen much more markedly when considerable doses are given, the lowest temperatures recorded having been observed in drunken persons, whilst animals exposed to cold have their temperature depressed to an enormously greater degree when they are alcoholised than when they are not so treated. For example, the temperature of a normal rabbit exposed to cold only falls about 2·8° C., whilst the temperature of an alcoholised rabbit may fall 19° C. In the same way the fall of temperature of a normal guinea-pig exposed to cold is only from 0·1° to 0·2°, whilst the fall in temperature of an alcoholised guinea-pig, similarly exposed, may be as much as 10°. These facts are of considerable importance in connection with conditions of susceptibility. Pasteur found, for example, that by lowering the temperature of a hen he could render it much more susceptible to anthrax infection, and other observations of an analogous character have been made. This and other points are worked up fully by Dr Laitinen, but this short sketch will suffice to indicate the scope of his work and the results he obtains.

Let us consider the relation of alcohol to phthisis alone. We have direct evidence that alcohol even in small doses predisposes to infection by the bacillary virus of this disease; and if this be true as regards primary infection, can it be any less true in regard to secondary and continued infection? The extension of the tuberculous process is merely an infection and invasion of other and surrounding tissues, an infection that we are constantly trying to cut short or prevent; and if alcohol increases the susceptibility of the tissues of animals to this infection, or interferes with a production of immunity which we are constantly trying to induce, it can scarcely be good as a permanent medicament or alimentary substance for human subjects suffering from this disease. Beyond this, however, there is another factor which should never be lost sight of. Alcohol, as we have so often been told, "insists" on being oxidised when it once gets into the body, and in phthisis pulmonalis, where there is such marked interference with the respiratory processes, and where in consequence there may already be an interference with the oxidation of the carbohydrates and fats, this may be a most serious factor in bringing

[1] Schäfer's "Text-Book of Physiology," 1900, vol. ii. p. 53.

about an accumulation of fat in the liver, and in setting up fatty degeneration in the cells of this and other important organs. I shall not attempt to describe the pathological changes set up by alcohol, but I should like to point out that fatty and fibrous changes are not confined to the liver in alcoholics, although they are most frequently associated with that organ. In this connection it must be remembered that one of the difficulties encountered in any attempt made to determine the pathogenetic action of alcohol is the very wide general toxic effect it exerts on the system. For instance, Mitchell Bruce[1] calls attention to the fact that the direct effects of alcohol on the heart and vessels are so "complicated with the many indirect effects which it produces on these organs, by deranging the functions of alimentation and assimilation, the nervous system and the kidneys, and with the secondary effects on the vessels and heart of chronic nephritis, due to the same cause, that it is difficult to determine which are direct and which are indirect." He is satisfied that dilatations of the heart and alterations in the myocardium are often met with as the direct result of alcoholism —acute dilatation, fatty degeneration of the myocardium being of frequent occurrence amongst alcoholic patients. In the post-mortem room my observations led me to very much the same conclusions: that there are two sets of conditions which appear to be the direct result of the use of alcohol. I have seen two cases — both in young, otherwise healthy people, who had succumbed to acute alcoholism—a condition of acute myocarditis without any endo- or pericarditis, in which there was cloudy swelling of the muscular fibres in a marked degree, in one this being accompanied by a peculiar hyaline condition of some of the fibres. In both there was marked interstitial change, resulting in the accumulation of numerous cells—leucocytes and proliferating fixed connective tissue cells—between the muscle fibres.

In the more chronic alcoholic cases, so frequently met with, fatty degeneration of the heart muscle, sometimes of a most marked type, is usually found; but, in addition to this, an increase of fibrous tissue between the bundles of muscle fibres, and in certain cases distinct evidence of muscular atrophy, may be recognised. It is in young alcoholics, and especially in those who have been otherwise healthy, that the study of these changes in the heart should be carried out; as in later life the changes in the blood vessels associated with Bright's disease, with cirrhosis of the liver and changes in the nervous system, conditions of malnutrition, many of them the direct results of alcoholic poisoning not always directly traceable to this condition, make their appearance.

[1] "Lettsomian Lectures on Disease and Disorders of the Heart and Arteries in Middle and Advanced Life," *Lancet*, London, 1901, vol. i. p. 845.

But the heart appears to be affected by alcohol long before any organic change can be made out in its tissues. For proof of this we must refer to the physiologist, who tells us that alcohol renders systolic output incomplete,[1] increases the diastolic pressure and the dilatation of the heart. He quotes Stephen Hales, who says: "The heart being in this case like a water mill that is surcharged with a flood both before and behind its force, must needs abate and grow languid." In this condition, experimentally produced on animals by the exhibition of alcohol, the heart may become so large that it can no longer beat in the confined fibrous pericardium, and finally cessation in diastole takes place; alcohol certainly lowers the arterial tension.

Perhaps the best tissue in which to study the direct action of alcohol on protoplasm is the nerve cell. It appears that alcohol is only one of a large group of substances which exert a definite and appreciable action upon these cells—acetate of lead, arsenic, bromine, antipyrine, cocaine, strychnine, toxines of rabies, tetanus, etc.[2]

Heinrich Dehio[3] carried out a series of experiments on the influence of acute alcoholic poisoning on nerve cells. These were subsequently repeated by Colin C. Stewart,[4] who, injecting alcohol subcutaneously into cats in quantities sufficient to produce acute alcoholism, found that Nissl's granules gradually disappeared (the protoplasmic basis becoming slightly stained) from the nerve cells of the cerebral cortex, from Purkinje's cells, and from the multipolar cells of the spinal cord, all this indicating a marked toxic condition, such as is met with in connection with diptheria, tetanus, and other poisonings, the changes being more marked as the dose of alcohol is increased, and as it is allowed to act for a longer period.

That such pathological changes could be demonstrated as the result of acute alcoholic poisoning, has only recently been made out, though every physician who has had cases of alcoholic poisoning under his charge could not but be convinced of the presence of some serious nerve changes in this condition. In regard to chronic alcohol poisoning, the conviction that alcohol acts detrimentally on the nerve cells and their surroundings and continuations, has not been nearly so widespread amongst medical men generally; but amongst pathologists, and especially amongst those working in our asylums, the number and variety of lesions met with as the outcome of this form of poisoning has been recognised and studied for some time past. Berkeley[5] quotes Bevan Lewis, one of our earliest, best, and most accurate

[1] Leonard Hill, Schäfer's "Text-Book of Physiology," 1900, vol. ii. p. 53.
[2] Schäfer, *op. cit.*, vol. ii. p. 604.
[3] *Centralbl. f. Nervenh. u. Psychiat.*, Coblenz u. Leipzig, 1895, N. F., Bd. vi. S. 113.
[4] *Journ. Exper. Med.*, N. Y., 1896, vol. i. p. 623.
[5] *Brain*, London, 1895, vol. xviii. p. 475.

observers, who says: "In alcoholic brains the cells become granular and show a shrunken protoplasm-stunted processes," which changes he maintains are "the after-effects of long-continued malnutrition and irritation of the cells by the poison." Following up these observations, Berkeley himself demonstrated serious pathological changes in the nerve cells and their processes as the result of chronic alcoholic poisoning. He found that in the cortical cells of the cerebrum changes occur in the nuclei and nucleoli, the former becoming irregular in outline, the latter becoming enlarged. Both these changes are met with when the cells are over-stimulated, or when they are badly nourished, and especially when there can be demonstrated any accumulation of waste products in the protoplasm of the cell, or in the lymph spaces of Obersteiner. Under the same conditions, marked changes take place, as we have seen, in the protoplasm of the body of the cell; but, in addition to these, the special processes of the pyramidal cells, and the special dendrites, also undergo alteration, assuming a peculiar moniliform appearance. All these changes are described by Berkeley, and figured with great clearness; and moreover, he has been able to correlate symptoms and nerve lesions in a most instructive fashion, and to associate the lesions with causes which may be grouped under the heading of overwork and fatigue. He points out that in vascular disorders of the brain, induced by the use of alcohol, the nerve cells are irritated by the presence of a poisonous stimulus and an insufficient supply of proper nourishment; they overact for the time; and then, as nutriment is still withheld from them, altered metabolism results. He, like Bevan Lewis, maintains that the beginning swellings of the dendrites of the sensory motor region is accompanied by "anæsthetic and paræsthetic symptoms, those of the purer and sensory regions by visual and ocular troubles and some amnesia, especially for recent events; or, in other words, cells that have the function of evolving thought cannot work properly, and defective memory results. Later, as the motor cells become more and more involved, and nuclear changes begin, continuous tremor becomes apparent, the muscles no longer co-ordinate perfectly, unless for a moment, under the direct influence of the will; still later, when a portion of the cell structures become highly degenerated, and the altered cells have become more numerous, the already tottering will power becomes less and less active, judgment and memory fail, and when the degenerative process has far advanced, an incomplete dementia is the final result."

Here is no speculation; the observers noted the symptoms in the alcoholic patients, followed the cases carefully during life, and examined them equally thoroughly and carefully after death. The conditions then found may be compared with those met with in healthy brains, in brains of animals over-stimulated

and exhausted, and with brains from patients suffering from the results of other diseases and poisonings. Moreover, Berkley[1] points out that the lesions produced by him in the rabbit may be met with in the human brain,—the same degenerative process, "varying only in degree; in some intense, in others less so." "Such lesions of the neuron are not," he again writes, "peculiar to the effect of alcohol, but may be reproduced by any irritant drug or bacterial toxic product circulating in the blood, and acting for a considerable time on the living protoplasm of the nerve cell"; and, finally, "alcohol which was supposed to be the least deleterious of all the series, exerts a very definite and destructive effect upon the nerve cell." Other observers corroborate these appearances in a most remarkable fashion. Vas[2] says that alcohol, acting on the great cells of the anterior horn of the spinal cord and spinal sympathetic ganglion, brings about a loss of their chromatin structure; the finely granular character is perceptibly diminished, and there is homogeneous swelling; the protoplasm becoming more darkly stained than in the normal cells; and Andriezen[3] describes the extensive changes in the neuro-dendrites and the neuraxons of the nerve cells of the cortex, the alteration beginning in the fine bead-like contact granules upon the substance of protoplasmic extensions; then moniliform swellings are seen "along the course of the terminal protoplasmic twigs." "With the further progress of the lesion, these softened and enlarged protoplasmic masses form irregular botryoidal masses, mainly clothing the now irregularly bared protoplasmic stem, which rapidly becomes attenuated." The changes in the cell body are of a similar nature, and consist in a gradual disintegration of the cell after the apical and other of the processes have become affected. Here and there, at the side of the cell body, the protoplasm seems to become frayed or, as it were, eroded. In other cases the cell protoplasm becomes vacuolated from within, and the process continues until the whole interior protoplasmic structure is channelled by holes and seams of liquefaction. In the descriptions and demonstrations given by these three observers alone, we have materials on which to found a study of the pathological changes set up by alcohol. The conditions under which these changes are produced are now more fully understood; they may in certain measure also be controlled, and we are thus in a much more satisfactory position or coign of vantage from which to study the direct changes that take place in these and other tissues; and we may ere long obtain still more definite information on many of the points around which such keen controversy is now carried on.

[1] *Loc. cit.*, p. 295.
[2] *Arch. f. exper. Path. u. Pharmakol.*, Leipzig, Bd. xxxiii. S. 141.
[3] *Brain*, London, 1894, vol. xvii. p. 669.

Leaving the nerve cells, and turning our attention to the nerve fibres, we find that the physiologists have attacked the problem of the effect of alcohol on the functioning of nerves. Gotch [1] indicates that alcohol, amongst other chemical anæsthetics, can diminish the functional attributes of living nerve fibres without causing permanent impairment, and can exert this effect when reaching the tissues through the blood. At first there appears to be a slight increase in the local excitability of the nerve, and, in a less degree, of conductivity. Very soon, however, in place of this excitation, the conductivity of the nerve becomes impaired, and this goes on rapidly before the local excitability becomes diminished. When the exhibition of the alcohol is discontinued, the normal conductivity is gradually and very slowly reinstated, but the excitability returns much more rapidly. It is evident that if this anæsthetic action be repeated for a few times only, no permanent change is induced, even in the functioning of the nerve; but from what has been observed of the clinical history of, and pathological lesions met with, in certain cases of peripheral neuritis, we should be quite prepared to find that even here the jar may go to the well once too often. In connection with this let me quote what Professor Schäfer says concerning alcohol and metabolism [2]: "It cannot, in fact, be doubted that any small production of energy resulting from its (alcohol) oxidation is more than counterbalanced by its deleterious influence as a drug upon the tissue elements and especially upon those of the nervous system."

May I be allowed to sum up my own opinion of alcohol in a few words. It is a narcotic poison, of which the pernicious effects are to be seen at all times and on every hand. It is a drug which, under certain conditions, *may* be valuable, but it is a dangerous medicament in the hands of any one but a physician; and even in the hands of the physician or surgeon, its exhibition is attended with dangers that attach to the prescription of no other substance in the pharmacopœia; these dangers are not moral only, but physical dangers, resulting from the action of alcohol on the tissues generally, but especially on those of the nerve centres. Its food *value* under ordinary conditions is practically *nil*, and, put in the most advantageous light, can only be temporary, and then of an extraordinarily slight and, I believe, wasteful character.

Probably during the discussion a number of other points will be raised, but for the present I should like to insist that in looking at the question of alcohol and its use in disease we are not justified in throwing aside entirely the results of experiments carried out with doses of alcohol which are not sufficient to produce intoxication, but are in proportion to those given

[1] Schäfer's "Text-Book of Physiology," vol. ii. p. 492.
[2] *Ibid.*, vol. i. p. 882.

every day to patients, and which go to show that whenever a virus produces a disease it acts more certainly and readily in an alcoholised than in a normal animal; when it is attempted to produce immunity in an alcoholised animal one fails, in many cases, absolutely; it is certainly always more difficult to produce it than in the normal animal.

PROFESSOR T. R. FRASER said — I regard it as a great privilege to be asked to take part in the consideration of this subject, and to be permitted to join with other members of this Society in showing our appreciation of the kindness of our friend Dr Woodhead in coming here to-night to express his views on a subject to which he has given much attention, and which is always one of great interest.

Notwithstanding that so much has been written and said on the subject of alcohol, and from many standpoints, as that of the pharmacologist, the physician, and the social reformer, I am sure I shall have your approval, Sir, and that of everyone present, when I say that we have listened to Dr Woodhead's paper with the keenest interest, and that, whether we coincide with or differ from any of the statements and conclusions he has advanced, we are at one in admitting the ability of his exposition.

That there should be differences of opinion is only to be expected, when we recollect that this substance, alcohol, is by some held to be of the highest value, by others of no value, and by still others to possess interest only as a poison.

This last quality must be fully and unreservedly recognised; but it does not exclude the existence of value in several directions, such as the therapeutical and even the social. The toxic properties after all are merely those which are to be placed in the same category as the properties of 60 mins. of croton oil when contrasted with those of 1 min.; or of 10 lbs. of beef steak when contrasted with 1 lb. The knowledge of the effects of the larger quantities is of much value in warning us against administering them, but it is obviously of no value in indicating the great medicinal and food benefits which may be obtained from the smaller quantities.

I cannot help thinking that some of the confusion in statements, and much of the difference of opinion regarding the value of alcohol, may be explained by insufficient discrimination of the effect of different quantities, by want of sufficient attention to the all-important question in pharmacology, and therefore in therapeutics, of quantity or dose. According to the dose, alcohol, for instance, may be regarded as a paralyser of the circulation or as a stimulator of it; as a hurtful or a beneficial agent in primary digestion; as a food substance to be treasured and used with trustful discrimination in emergencies, or only

as a disreputable poison to be shunned by all self-respecting persons.

As I have thus incidentally referred to three actions which alcohol is claimed to exert, which from the standpoint of the medical practitioner are the most important, they may be used as illustrations of the great importance of clearly distinguishing between the effects of small as contrasted with large quantities; and by a large quantity I mean not only a single large administration, but also a continuous administration over a considerable interval of time of a succession of single small or moderate doses.

The first of these actions, that upon the circulation, probably constitutes the foundation of the very frequent application of alcohol as a so-called stimulant. Is this application justifiable? Were we only to have regard to the effects of large administrations the answer would undoubtedly be in the negative. When, however, we have regard to the changes in the circulation following the absorption of relative small quantities, I believe that the experimental evidence is absolutely in confirmation of its frequent therapeutic use.

Such quantities, by a direct action on the heart, undoubtedly increase the rate and force of its contractions. It is a very rapidly absorbed substance, and, therefore, the effects are quickly produced, though they are not very lasting. Further, as I and others have convinced ourselves, this stimulant action may be produced in a healthy person if excessive quantities be avoided without the slightest evidence being obtained of an after weakening or paralysing action on the heart. The heart merely returns to the former standard of its activity.

Not only is the circulation stimulated by this direct action on the heart, but it is stimulated also by an action on the blood-vessels. Quantities sufficient to increase the rapidity and energy of the heart's contractions also slightly and temporarily contract the blood-vessels, and if the quantities are increased only a little more the heart contracts still more effectively, but the blood-vessels dilate; and thus the distribution of blood is accelerated and widened, and the functions of many organs and structures are increased. There is produced not only a stimulation of the circulation, but a stimulation so widely extended that alcohol, more than almost any substance, may, though in somewhat inexact language, be regarded as a general stimulant.

The gain to therapeutics of this action alone is obvious, not only where the heart is weak, but also where the blood distribution is imperfect; and it not only itself justifies completely the inclusion of alcohol among our therapeutic resources, but, I venture to think, reduces the usefulness of the medical practitioner who refrains from using alcohol in such circumstances as I have indicated.

If we now turn to the second action I have mentioned, that on digestion, it may again be stated that large quantities paralyse digestion; but, on the other hand, that small quantities improve digestion.

Such quantities increase the flow of saliva without impairing its digestive properties; they augment the secretion of gastric juice, and actually enhance its peptonising power; they greatly stimulate the peristaltic movements of the stomach, and so increase the efficacy of the abundantly secreted pepsin and acid; and they apparently cause leucocytosis, and may thus further the carriage to all parts of the body of albumin, which has been absorbed into the lymph spaces in the stomach walls, very much in the same way as the red cells distribute oxygen widely; and thus both primary digestion and nutrition are improved.

In regard to the direct effects on nutrition of absorbed alcohol, the proposition may without hesitation be stated that large quantities impair or suspend it, probably by paralysing the activities of all cells in the body. On the other hand, small quantities further nutrition and serve the purposes of food by supplying the body with energy.

Again I must content myself with stating conclusions without attempting to describe fully the experimental and other data on which they are founded.

I would remind you that after the injestion of such quantities as are useful, only an insignificant percentage is eliminated unchanged from the body and that oxygen absorption and carbonic acid elimination are increased. Either or both of these facts is sufficient to show that alcohol is consumed in the body within certain limits, and, if consumed, it must of course evolve energy which supplies force, and so alcohol acts as a food. Its food value, roughly, is equivalent to that of an equal weight of a fat such as butter, and while it appears incapable of modifying the nitrogenous metabolism, it can replace carbo-hydrates and economise the fats of the body.

As a food, further, it has the advantages in disease of making no demands on primary digestion, and of being very rapidly absorbed and diffused. But these are not the only facts in support of the proposition that it has some value as a nutritive substance. It has also been shown to be capable of maintaining the equilibrium of nutrition in normal persons purposely restricted to an insufficient dietary; and there is much clinical experience that it can do so, for short periods of time, in conditions of disease, when both primary and ultimate digestion are paralysed and impaired.

At the same time, I fully concede that in healthy conditions, alcohol is a wasteful and expensive food and one attended with moral dangers. In conditions of disease, however, where the

stomach is unable to retain or to digest other foods, it may be, and every day is, used as a nutriment, and is then found capable of easy absorption, without any process of digestion being required, and capable of sustaining life for brief periods, not only because it supplies energy, but also because it stimulates a flagging and a failing circulation sufficiently to allow blood to be conveyed to expiring organs and tissues.

In many of these urgent conditions, another action to which I have not made any reference, that on body temperature, is often produced with much advantage. The temperature is reduced by moderate doses because of increased heat dissipation and radiation from the body, and by large doses, to a still greater degree, because such doses not only further heat dissipation, but also reduce the activities of probably all the cells of the body. It is thus an antipyretic, and clinical experience has produced evidence in favour of alcohol in many stages of fever.

There is another action of first importance, that on the central nervous system, which I purposely refrain from discussing, as I know it will adequately be considered by Dr Clouston, who is to follow me.

I have said enough, however, to show that alcohol possesses a singular number of actions of value in disease. There is not generally only one problem, but several problems before us when we treat a patient. It is sometimes the cardiac feebleness. It is sometimes the impaired digestion. It is less frequently the requirement to sustain life over a grave crisis, and even in that circumstance we may succeed in preventing death, not only by stimulating the circulation, but also, I feel assured, by liberating the energy produced by the combustion of alcohol when it is administered as a food.

Before I sit down, let me add, in reference to Dr Woodhead's remark that there exists a certain amount of experimental evidence that alcohol increases the infectivity and virulence of pathogenic organisms, that this action does not appear to be a powerful one, at least in the absence of extreme immoderation in the use of alcohol. I have had occasion to consider the incidence and mortality from the infective disease, plague, now raging as an epidemic in India. The Natives of that country, who by religion and circumstances are largely abstainers from alcohol, have shown a case-mortality in the Bombay Presidency of nearly eighty per cent., and in some places in India of even upwards of ninety per cent. On the other hand, the case-mortality has been only about twenty-nine per cent. among Europeans in the Bombay Presidency, although they use alcohol as commonly as Europeans elsewhere. In the recent outbreak in Glasgow, where the cases occurred for the most part among those living in the insanitary districts of that city, where immoderate indulgence in alcohol is only too prevalent, the case-mortality was only

twenty-eight per cent., or much the same as that of the European in India. It is to be noted, further, that the evidence points to the fact that the strong and well-fed are as susceptible to the disease as the weakly and under-fed, and therefore that the relative immunity of Europeans in India, no more than the liability of the Europeans in the poorer districts of Glasgow, cannot be explained by physical strength or good feeding. I do not, of course, draw the inference that alcohol exerts a protecting influence, but only, as I have already said, that if, in any circumstances, it increases liability, its power of doing so can only be insignificant.

DR CLOUSTON—In regard to the effect of alcohol upon the higher nervous system, one fact is now quite recognised, which is that in the production of the most serious of all nervous diseases—mental disease—alcohol stands as the highest of all exciting causes, being responsible for from 20 to 25 per cent. This by itself would be a very grave fact, but a statement of this kind required some explanation in order that it might not be misinterpreted. When they considered the neuropathic brain, which in the vast majority of cases was the real and original producing cause of mental disease, it would be recognised that if alcohol did not act as a cause, a great many other causes might come in to upset the mental balance; also, this taking to alcohol in excess was in many cases a symptom rather than a cause. There were facts which showed this very well. In certain counties in England there was a good deal of quiet beer drinking, and where spirit drinking did not prevail. In these counties alcohol was put down as a factor in the causation of insanity to a much less extent, and yet they produced as many lunatics as did Edinburgh. Another extraordinary fact was that the Society of Friends was the most sober and self-restrained of all sects, yet mental disease prevails to as large an extent amongst that Society as amongst the general population. The Quakers, no doubt, have inbred to a large extent, but these facts show that the figures as to alcohol must be conditioned in many ways. When they considered the quality of brain on which alcohol has the most pleasant and fascinating effect, they found it was that brain in which the first action of alcohol is to diminish the great mental quality of inhibition, and at the same time to arouse intense desire. They found that many such brains were the most sensitive of all human brains—and this covered the whole field of the poetic and artistic tribe. On this quality of brain alcohol was the most potent and destructive of any toxin that could be applied. There was no doubt of the intense pleasure which alcohol caused to such brains; and he hoped that they had all to some slight extent the sensitiveness which made men capable of such enjoyment. Dr Woodhead might

say this was all very well—that while alcohol was producing this intense enjoyment, it was no less exerting its destructive influence. There was no doubt, he feared, that human nature being what it was, the balance of choice would always tend towards pleasure, and towards raising an ideal in life, and so long as no destructively evil effects resulted—that is, so long as a reasonable moderation was practised—the ethical sense of mankind was not disturbed. This was a side of the question too little taken into consideration by those who point out the evil effects of alcohol.

In regard to the psychological action of alcohol, a number of experiments of a most careful kind had lately been made by Professor Kraepelin. German students were tested in a scientific way as to certain mental powers and reactions before and after they had fixed doses of German beer. There was no doubt that the full effect of small doses was to liberate energy. On the higher brain cells there was a certain katabolic effect, and this was accompanied by a mental or emotional pleasure; but, at the same time, the cognitive and reasoning output was undoubtedly of a poor sort—it was not so good in quality and accuracy as if cell katabolism had taken place in a normal manner. Larger doses resulted in a slower and worse output all round. But there was one very important result which they could not too constantly keep in mind. It was found that in these experiments, that after these students had thus taken alcohol for a few days and then stopped its use, it had a much greater effect, in the same direction, when resumed. The more confused quality of output was then produced with only half the dose of alcohol that acted before. This was entirely consistent with experience. They knew that after the drunkard had become addicted to the intemperate use of alcohol, it produced its effect more easily. Some molecular change in the brain cells was left, through which they were more irritable and explosive.

They had had Royal Commissions on many subjects, and it seemed rather extraordinary to him that, so far as he knew, no scientific commission of experts had been ever appointed to investigate this great medical and social question from a purely scientific point of view. It affected the general health of the community, and was certainly worth inquiring into. There was no doubt that, speaking generally, those unaccustomed to weigh the value of experiments were confused between the statements made by the total abstainers on the one hand, with the enormous body of facts which they have at their back, and by the statements made by those who considered the moderate use of alcohol legitimate, on the other. He believed that such a commission might, with our latest knowledge of the effects of alcohol on mental disease, on nervous function, on general disease, and on social and economic questions, lead to very

valuable results. He could not conceive a better outcome of the discussion than that their chairman should take steps to induce those in power thus to look into this question.

There was another practical question to which he would refer. In the treatment of admitted alcoholics they all knew that one of the great difficulties is that the patients cannot help in their treatment sufficiently themselves; and they all knew that some legislative help was needed in these cases to enable us to treat them effectually. Should not the whole profession urge that such a legislative measure is needed? Surely this would not be too much for the legislature to grant.

Lastly, he would be very sorry to sit down without saying that, as a result of his own experience, the effect of the excessive use of alcohol on the higher functions of the brain is an extremely terrible one. Taking a broad view of the customs of society, of the prevalence of the neurotic or neuropathic brain, and of the extraordinary prevalence of the ignorant abuse of alcohol, he thought that they, as medical men, should endeavour to make their opinion felt by the public; and that as a profession, by an emphatic and general expression of their opinion, they should show that the excessive use of alcohol was one of the most terrible ailments of modern society; and that more especially was it harmful in our city life. He believed that such a pronouncement, which should be reiterated at stated intervals, would do a great deal of good.

DR AFFLECK might be regarded as one having strong views upon the question, which he thought had been tested by experience. His opinion in respect to the administration of alcohol was that while he should not like to be without it, it should be regarded very much as an emergency remedy. Though the statements made by Professor Fraser were very interesting, he thought they were rather dogmatic and some of them open to serious question. With regard to his remarks about the relative immunity from plague of those who took drink, he would ask Professor Fraser, when he gets a case of pneumonia into his ward and is told that the man is a drunkard, if his prognosis is not a very serious one. He would often find that the patient died, not from the extent of lung involved, but from the overwhelming character of the poison he had imbibed—his chronic alcoholism put him in an unfavourable position for fighting the disease. Alcohol had been very much used in the treatment of pneumonia, and had been recommended to be given in very large quantities; and while he did not deny that it had its uses, he had been grievously disappointed in regard to it. He had rarely seen a patient assuming a serious condition who was benefited by the large doses recommended to be given. With regard to the use of alcohol in other than emergency cases

—as an aid to digestion for instance—he thought a terrible danger lay there which they should not fail to recognise. What Professor Fraser had said about its action might be true; but if it had to be called into use every day was it likely that its alleged good effects would continue for a length of time; and even granted that it did exert a beneficial effect might it not be too dearly purchased by producing tissue changes in the body which would make themselves manifest in the later period of life. He thought the evidence of *post-mortem* rooms would bear him out that the effect of habitual small doses was very often disastrous. In conclusion he would say that he felt himself more and more chary in ordering alcohol—he felt a great sense of responsibility in ordering it knowing how dangerous a thing it was, and they could not dissociate the moral aspect from the medical aspect of this question. There was no remedy which was so frequently suggested to medical men—they were invited and even coerced into ordering it. This was a terrible temptation to young practitioners, and it required a pretty strong man sometimes to say to patients' friends that they would be better without alcohol at all. He held that unless the practitioner conscientiously believes that the patient needs alcohol he should not order it.

DR W. LESLIE MACKENZIE considered the public health aspects of the question, and asked:—

1. *How is Alcohol related to Public Health?*

In two ways: first, directly as a factor in the ordinary food environment; second, indirectly as a factor in the moving equilibrium of the social organism. In its direct action, it may produce a series of fatal diseases; it increases the fatality of many other diseases; it predisposes to some. Predisposition and fatality are alike preventable, because both are due to a removable cause. In its indirect action, it lessens the sense of social responsibility in every grade, from the family upwards to the race; it tends to generate neglect of routine; it thereby impedes the course of social development; it predisposes its victims to satisfaction with a lower standard of living; it may produce economic inefficiency; it thus generates a lower standard of comfort, and a lower standard of cleanliness; it ends in aggravating the conditions that foster many of the communicable, and many of the non-communicable, diseases. Doubtless, there are many counteractives. What I have said is true mainly of those persons only that, under stress of bad economic conditions, exceed in their consumpt the commonly accepted normal of two ounces of absolute alcohol in the twenty-four hours. But in lesser degrees, down to the consumers

of a small undefined amount, the same propositions are more or less true. But at the lowest end of the social scale, as at the highest, there are counteractives. When alcohol has exhausted the drinker's money and credit, it conducts him to the poorhouse, where he becomes as sober and almost as useless as he formerly was drunk and incapable. His family, too, are more or less well guarded from disease, and moderately well nurtured into efficiency. At the highest end of the social scale, the counteractives are better food and the good offices of friends. Both by the segregation of the lower and the protection of the higher victims, the death-roll is every year robbed of a few names. And there is something positive to say for the four-ounce-a-day drinker. He lives in a state of persistent mental erethism; he is never drunk; but he suffers from slight disturbance of perceptions; he exaggerates the facts favourable to him; he does not appreciate the facts against him; he spends his business days in a state of mildly exalted feeling, of which the mathematical limit is universal defiance or fighting-fitness. To men in this erethistic condition, life is less of a worry than to the absolutely sober; the wear and tear from alcohol is more, but the wear and tear from circumstances is less, and we must, I think, allow something positive for the habitual inconsequence of such a life. My observation of many classes of men in Leith has tended to confirm this suggestion. It is worth noting, though it may be a pure accident, that while "Edinburgh has one licensed house to every 378 inhabitants, Glasgow one to every 401 inhabitants, Paisley one to every 392, Dundee one to every 369, Aberdeen one to every 367, and Greenock one to every 358," Leith, whose death-rate is notoriously lower than the death-rate of the others, has yet one licensed house to every 274 inhabitants. (Rowntree & Sherwell, Pop. Ed., p 29.)

I have stated the case in the most abstract way; and it is impossible to exhaust even a small part of the diseases correlated with alcohol. I confine myself to the connection of alcohol with the infections.

2. *Does Alcohol Predispose to Infection?*

This question cannot be answered by a simple "yes" or "no."

Infectious diseases are, for the most part, diseases of early life, when alcohol as a factor is practically of no account whatever. Diphtheria, scarlet fever, measles, whooping-cough, chicken-pox, mumps, German measles, belong predominantly to the non-alcoholic ages. It is possible that the use of alcohol in their treatment may do more harm than good—that is a matter for clinical investigation; but it is certain that alcohol has no direct part in predisposing the constitution to them. The soberest child born may at any moment take any of them. And

I am not aware of any proof that the children of mothers alcoholised during pregnancy are any more susceptible than others. It may, I think, be taken as established that, with a small exception for idiosyncrasy, all children are, in greater or less degree, susceptible to the diseases named. Alcohol, therefore, as a predisposing factor may be excluded.

Is the same true of the other major infections? I think, on the whole, it is. Enteric fever, small-pox, typhus fever, erysipelas, septicæmia, pyæmia, croupous pneumonia, cholera, plague, yellow fever, malaria, beri-beri, tuberculosis, syphilis, and some others are—given the necessary exposure—as, with one or two exceptions, ready to attack the non-alcoholised as the alcoholised. One cannot say that, as a directly predisposing factor, alcohol plays any special part. It is at once admitted that, where there has been alcoholic excess, or persistent alcoholising without special excess, the prognosis is bad, particularly in pneumonia, typhus fever, and perhaps in the many varieties of septic disease. But with these exceptions I do not think it can be shown that alcohol plays any considerable part either in creating a predisposition to them or in increasing the total mortality from them. All that we can safely say is: Given a bad alcoholic history, the case mortality in certain infections will be higher. But against this we have to say also that a bad alcoholic history is not generally established until after 40 or 45, and then the physiological susceptibility to most infections is less; the exposure to them is, as a rule, less, and in many indirect ways the organism is better guarded.

To take, now, one or two diseases individually.

Typhus.—Typhus is not specially a disease of the alcoholic, but it is certainly more fatal in those of alcoholic history. Is this a hygienic loss or a hygienic gain? It is both according to the standpoint. If we wish to preserve the alcoholic, it is a loss; if we wish to eliminate him, it is a gain. In neither case does alcoholism affect the spread of the disease, and that is the cardinal fact for preventive medicine. But typhus has itself been practically eliminated from Britain at least. Consequently, its contribution to the death-rate has ceased to be important.

Cholera.—The broad fact that cholera prevails in India among races that consume practically no alcohol, and does not prevail specially in Western European countries where alcohol forms an ordinary article of diet, is enough to justify us in discarding alcohol either as a predisposing factor or as seriously increasing the case death-rate. In certain parts of Russia cholera is practically endemic, and alcoholic excess is very common; but the prevalence of the disease may be due as much to economic conditions as to alcoholic excesses. Not that in cholera a bad alcoholic history is other than unfavourable; but cholera does not so discriminate between the ordinary

four-ouncer and the under-two-ouncer as to make an appreciable difference in the comparative totals.

Plague.—The same argument applies to plague. At no stage can it be said seriously to be influenced by alcohol. It attacks non-alcoholic races equally with the alcoholic.

Croupous Pneumonia.—Here probably alcohol predisposes both directly and indirectly; it certainly affects for the worse the prognosis. Pneumonia would, of the microbic diseases, probably profit most by the abolition of alcohol from the environment. The other infections would not benefit much directly by the abolition of alcohol. This is to say that, so far as infection generally is concerned, the abolition of alcohol is not, to any appreciable extent, a direct preventive. An indirect preventive it certainly would be, for we have overwhelming evidence that the absence of alcohol means improved social efficiency, and, within limits, improved economic conditions, and these in turn coincide with an improved standard of health, and consequently with an improved sanitary environment — less overcrowding, better nutrition, and greater cleanliness. When I say "within limits," I mean to indicate my belief that alcohol is in part a cause, but in part also a consequence, and although the more sober nations are likely to out-distance the less sober in the production of wealth and in elaborating the methods of its distribution, the total abolition of alcohol, or the total cessation of drunkenness, will leave untouched the fundamental economic problems of our time.

3. *Is Alcohol in some degree a necessary and indispensable part of the food environment?*

This question may be more exactly put—Is alcohol, like water, or certain salts, a specific supply of a specific organic demand? Can a race exist without it? Is there a "craving" for alcohol as there is a craving for water?

Dr Archdall Reid maintains that there is; but the evidence he adduces is not to my mind conclusive. His argument that abstinence from alcohol permits the survival of a greater number born with this "craving" appears sound enough, if this "craving" be first demonstrated as a fact. What unquestionably, however, is true is that abstinence from alcohol increases an individual's susceptibility to alcoholic poisoning. The total abstainer, with a small exception for idiosyncrasy, answers always to a glass of whisky, and usually to much less. Consequently, it may be allowed that widespread abstinence creates in each generation, quite apart from questions of inherited diathesis, a large proportion of persons that are easily made drunk. It follows that a smaller consumpt per head will mean a larger percentage of temporary intoxications. This is seen in the six-monthly experiments of our rural fairs, where

a very little whisky serves to line the roadside with young men drunk. I am inclined to explain in the same way part of the increased police returns. It may be that an increase of drunks and incapables is an indication of the gradual supersession of chronic soaking. The chronic drinker is not usually incapable enough to come before the police magistrate.

But this increased susceptibility induced by abstinence does not imply the creation of a craving. And even if it did, it is illegitimate to assume that such a craving is not amenable to any but a specific satisfaction. The acquired appetite induced by the use of a drug is a well-known condition; but it requires more proof than I have yet seen to show that alcohol specifically satisfies any primary organic appetite. The existence of the non-alcoholic races is alone enough to cast a doubt on the whole theory.

But if there be no primary appetite, alcohol cannot be said to be a necessary and indispensable part of the food environment. The transmission as such to the child of an appetite acquired by the father is an unproved assumption; the passage of alcohol from the parent directly to the germ is physiologically possible, but it has not, so far as I am aware, been shown definitely to generate a congenital specific craving in the child, however much it may disturb the development; the passage of alcohol from the mother to the fœtus may make the fœtus drunk from time to time, and so initiate a congenital craving, and this is all that the facts require. It is a question for proof whether such a specific craving is ever congenital. That this craving, acquired *in utero*, is transmitted to the third generation without alcoholic indulgence by the middle host, needs a vast amount of proving.

Each generation has to take care of itself, and I am not much moved by such possible dangers to posterity as the creation of more susceptibles is supposed to entail. History is strewn with discredited forecasts, and our knowledge of social laws is too elementary to make dogmatic prophecy other than unprofitable.

4. *What can the Public Health Organisations do to prevent the evil effects of Alcohol?*

Through the Food and Drugs Acts, they can exercise a certain very rough control over the quality of the alcohol sold for general consumption. That beer should not contain arsenic is certainly a gain; but is it equally a gain that whisky should not contain water? From what I have seen and know of the *Food and Drugs Acts*, I doubt whether, even if applied persistently and stringently, as in many places they are, they would to any appreciable extent lessen the evils even of bad whisky—whether by "bad" we mean over-watered or adulterated.

For the prevention of open drunkenness, the magistracies can do something. Leith, once more, has shown some good results. But these results are results for decency, not for public health. The stopping of open drunkenness will not much affect the production of acute alcoholism, diseased livers, and shattered nervous systems.

For the present, the Local Authorities for Public Health Administration have very few points of direct contact with the trade in alcohol. Legally they do not have any, and I do not think it would be much advantage if they had. The alcohol question is so much more than a question of public health that the little a Public Health Administration could do directly is better done by other organisations. Indirectly, the Local Authorities can do something, they can help to improve the housing of the labouring classes, and thus by improving the environment they can do something to harden the rising generation.

They could also do something for the food supply. They could collect, purify, and distribute milk. They could organise public services for the day nursing of infants, and so lessen the overwhelming home-burdens that too often drive the mothers to alcohol. They could institute wash-houses, which would relieve the home and improve its amenity. They could—and do—organise amusements; but of the ultimate benefits of the amusements organised, I have grave doubts. They are not always unassociated with alcohol; they encourage the scattering of attention on trivialities; and, for my own part, I do not believe that amusements of the gregarious order, where a man goes with men and leaves his wife to drudge at home, do much either to drain the alcoholic swamp or to strengthen men to wade through it. And for the dangerous ages—the climacterics of man and woman—amusements do nothing but evil. Indeed I am inclined to echo Sir George Cornewall Lewis's epigram: "Life would be tolerable but for its amusements."

Note.—Dr Archdall Reid has recently somewhat modified the extreme position referred to above.

DR JAMES RITCHIE thought that what they wanted was facts—facts pathological and physiological—not that he objected at all to laboratory experiments, some of which had great value. But laboratory experiments were conducted under peculiar influences, and were often surrounded by circumstances which did not occur in nature; and if they could get examples conducted under natural conditions and over a sufficiently wide area, facts deduced from these would be of much greater value. Now it so happened that some Life Insurance Companies had been in existence for a long time, and the results of their investigations were carefully prepared and tabulated. These

companies found that those who dealt much in alcohol were very bad lives. They found that the large extra rate of 20s. per cent. charged in the case of those dealing in alcohol was not sufficient. There were, however, certain companies which had two classes of risks—non-abstainers and total abstainers. The Sceptre Insurance Company was one of these, and recently made an investigation into the results of their work for sixteen years, up to 1899. They found that on the general side they had a slightly smaller number of deaths than the anticipation; but on the total abstainers' side they had 23 per cent. fewer than on the general side. The United Kingdom Temperance and General Insurance Company, which has a similar classification, had made investigations, and their experience was at each investigation somewhat similar, viz., on the general side—and of course the companies are particular about the habits of those they insure—the death-rate was 1½ per cent. below the expected rate; while on the total abstainers' side it was 26 per cent. less. He thought these were facts well worthy of consideration. There was another comparison. Among the working classes Industrial Insurance Companies insure also against sickness and accident; and it was found that among the Society of Foresters, who are not necessarily abstainers, there was three and a half times as much sickness per annum as among the Rechabites, who are total abstainers. Although the directors of insurance companies are not necessarily total abstainers, for policies which cover the risk of accident they take from total abstainers 10 per cent. smaller premiums than from non-abstainers. He was bound to conclude that, so far as duration of life was concerned, and so far as liability to sickness or accident was concerned, total abstainers are better lives than those who are supposed to take alcohol even in moderation. These were facts; but he might also allude to his own experience. In relation to gout, he believed that alcohol so modified processes going on in the body that it predisposed to gout, and that the gouty patient was very much better without alcohol at all. The text-books were at one upon this point he thought, but he questioned very much if medical men were not afraid to communicate this opinion to their patients. In regard to rheumatism, his experience was precisely similar. He believed that alcohol predisposed especially to chronic rheumatism. In relation to heart troubles, he thought that perhaps they got the best idea from cases of acute general dilatation; also from cases of chronic dilatation due to failure of compensation. He never gave alcohol to these cases; even when they had been in the habit of taking it, he immediately stopped the alcohol and put them to rest. He believed that not only was alcohol unnecessary in these cases, but that it was a distinct evil. Professor Fraser had said that

in small quantities alcohol stimulated the heart, and explained also its action in dilating the capillaries. This was also his own experience. But, on the other hand, he found that there was an exactly similar depression after. And what was the patients' experience? After a short time he desired another dose of stimulant, and with a feeble heart he went on stimulating at intervals with unfavourable results. He quite acknowledged that one might get distinct benefit by giving small doses of alcohol when there was a high tension pulse, but he believed one could do very much better by means of other remedies, and that alcohol should be kept for cases of emergency.

DR P. A. YOUNG showed some diagrams containing the results of the experience of the Sceptre Insurance Company in respect of its two classes of insurers—non-abstaining and total abstaining. This had been already alluded to by Dr Ritchie. He pointed out also the difference in death-rate between the Rechabites and Foresters, two societies drawn from the same stratum of the population—the industrial working-man—and who were very probably gaining about the same wage and had the same environment, except that the Rechabite was a total abstainer and the Forester need not be. At age twenty-five the death-rate was for the Rechabites five per thousand, and for the Foresters seven and a half per thousand, and a similar difference existed at other ages. It was also shown by the table that the Rechabite death-rate was lower than the average experience of twenty of the best British Insurance Companies, who, of course, always exclude drunkards.

DR FOULIS would like to ask—Does alcohol keep the heart going in those diseases where it has a tendency to fail, and where the disease tends to end in death?

Professor Fraser had made some extremely interesting statements, yet he was staggered by what he had understood him to say, that the effect of alcohol in small doses was to stimulate the heart, and that nutrition really resulted—that force was given as a result of the stimulation. His experience was the opposite of that—that the effect of stimulation was to reduce force. He had seen a man going along the street hilarious and happy, and in a short space of time he had to be carried to bed with his boots on. That was an illustration of what he meant, that stimulation is followed by exhaustion. He held the view that action was always followed by reaction, and had therefore come to the conclusion that alcohol is of very little if any importance in assisting the heart's action in what he might call chronic disease.

DR WELSH thought that Dr Woodhead had already pointed out

the chief lines on which pathological changes were met with, but these were almost entirely in cases where large doses and habitual doses of alcohol were taken. The present discussion had gone in what he thought was a more useful direction—on the effects of small or moderate doses. Referring to the effects of alcohol on himself, being very susceptible to its action, he found that a small dose in the middle of the day interfered with the more finely co-ordinated movements. In the early part of the evening also he found that power of mental work was diminished. The work did not bear the cold scrutiny of next morning. But there was another point to which he wished to refer specially. He found that a small dose of alcohol had a very beneficial effect in regard to insomnia, from which he suffered occasionally. He was surprised that no reference had been made to this effect of alcohol, as he had known elderly men who have relied upon this as a very important means of getting to sleep. Small doses had been taken beneficially for years by persons that he knew of.

DR CHURCH, in reference to giving alcohol *in extremis*, pointed out that it acts differently in disease to what it does in health. The only thing to do was to give small doses and watch carefully. If it increased an already accelerated pulse it was not to be given. If it has the effect of lowering temperature, lessening the heart rate, it should be continued.

DR CLAUD B. KER was greatly interested in Dr Woodhead's paper, because it had succeeded in shaking one of his fondest beliefs—that if there is any disease in which one depended upon alcohol especially—it was diphtheria. He was certain that the majority present would agree to that. He had, however, now learned that, at all events under certain conditions, not only is the person or animal more prone to take diphtheria, but that actually the action of the toxin is strengthened by the circulation of alcohol in the serum. As regards proneness to infection, he was inclined to believe it; but was bound to say that he would be very sorry to have to treat cases suffering from toxæmia without alcohol. Whenever he attempted to cut down alcohol in his diphtheria wards, he was very often confronted with sudden heart failure. It was about the only disease in which he used alcohol as a routine. In every other disease one judges as Dr Church had said by the condition of the pulse, the age of the patient, and the effect produced by small doses. But in diphtheria he must say that two out of the last three heart failures which occurred had been in cases which were not given alcohol. He had repented in these cases of not giving it. Now he wished to know from Dr Woodhead what he was going to give us in exchange for alcohol. While admitting that it might be toxic,

he did not see that, because it was a poison, it might not counteract a poison. He thought that if there was anything against the use of alcohol in diphtheria it was its tendency to lower temperature—that was the weak point. But what were they to substitute for it. They must not give strychnine—the patient would be dead while they were thinking what to do. They had learned a good deal during the discussion, but had not kept altogether to the practical side of the question.

PROFESSOR FRASER in replying thought that most of the objections raised had already been replied to. Dr Ker had in a very convincing manner pointed out the stimulative action of alcohol in certain emergencies or crises. There were phases not only in infective diseases, but in every acute disease, where the heart tended to fail, and then, as Dr Ker had said—what is one to do? Alcohol required no previous digestion; it was absorbed with great rapidity into the circulation; its effects were produced almost immediately. Looking at other substances; strychnine could not be substituted for it as it acts only on the blood-vessels—it does not affect the cardiac contractions. With any other substance, before its influence could be felt, the patient would probably be dead so far as our interference is concerned. He wished if possible to qualify some impression which had been produced by his remarks. He had at the outset confined himself to the effects of small doses. Large doses were certainly extremely harmful. If they looked upon alcohol as a food substance it must be regarded as having only a very temporary food action. Its operation was that of an insufficient food — only supplying certain elements, on which alone nutrition could not be maintained. Further, as a food it would be extremely expensive and wasteful; and its use was unfortunately attended with an enormous amount of moral failure. His position was rather to show that alcohol was a medicinal substance, with well defined medicinal actions—not to put it in the category of the effect of sixty minims of croton oil as contrasted with one minim, or of ten pounds of beefsteak as contrasted with one pound. His friend Dr Affleck also appeared to have misunderstood what he had said. In reference to pneumonia there again was the difference between small and large quantities or the aggregation of moderate amounts. Of course the prognosis of the alcoholic in pneumonia was greatly prejudiced. Although he knew alcohol to be a poison, he did not use it in such a manner as to produce a toxic action, and did not hesitate to employ it in judiciously adequate doses to produce stimulation—it may be to produce force by its combustion, and thus enable dying organs to revive, when the stomach is so disordered as to be incapable of digesting

food substances. He believed this was the cumulative experience of a large number of physicians.

Dr Foulis had also, he thought, misunderstood what he had said. The diagrams showed the effect on the pulse of a single dose of alcohol. Frequent tracings were taken, and at first there was shown to be a stimulation both as regards force and frequency; but gradually these effects were departed from until ultimately the pulse was restored absolutely to its initial condition. The observations were continued some time longer and there was no reaction below the normal point. So he therefore contended that alcohol could be used so that the reaction need not be so great as to produce a condition of greater enfeeblement than the original one.

PROFESSOR WOODHEAD in reply would like to say first how exceedingly glad he was that this discussion had taken place. He thought it had brought out a fact concerning which there is a good deal of doubt, or should he say confusion, in the public mind. They had heard very definitely from Professor Fraser that the use of alcohol as a drug refers to small quantities given on single occasions. He thought that was a fact which could not be made known too widely, because there is an idea on the part of many who are advised to take alcohol that it is not to be looked upon as ordinary medicine, and they go on taking it for long periods in the hope that the improvement would be repeated, when it should be confined to very limited occasions only. It was a very important point to bring out—that alcohol is to be used only on occasions of emergency. While on this part of the question he might reply to Dr Ker that he was not aware of having ever said that alcohol was not to be used as a drug. He believed with Dr Ker that, given a certain result to be obtained, one must give any drug in the pharmacopeia; and if alcohol is the only suitable drug, then Dr Ker and those working on these lines must use alcohol as a drug to gain such definite result. He was not against the use of alcohol under any circumstances. If a definite end is to be obtained it was the duty of every medical man to use it; but he maintained that something should be known of the end to be attained, and the dangers attending its use.

In connection with the action of alcohol being stated to produce leucocytosis, the experiments on animals were directly opposed to this—there was a distinct leucopemia. He would mention in connection with these experiments that they were not carried on for long periods only. The same results were obtained when alcohol was only given for a few days. In three or four days the exhibition of these small doses was quite sufficient to increase the susceptibility of the animal to the

action of the three poisons mentioned, but of course it was much more marked when the treatment was prolonged.

As regards the increase of metabolism, and the statement that the amount of carbonic acid gas thrown off was always enhanced by the administration of small quantities of alcohol, recent experiments rather go against this. Professor Fraser shakes his head; but he, the speaker, could only refer to results obtained during the last couple of years by those working on absolutely fresh patients—in these it did not always hold good.

As regards the rapid combustion of alcohol always meaning an increase of energy, there was no doubt a rapid increase of energy; but it appears, in certain cases at any rate, to be an explosion of a not very useful character. He was now speaking from an experimental point of view. It would be absurd of him to take up the position that what occurs in animals must also occur in the human subject. He only brought the matter forward that it might receive some attention from those considering the question.

The antipyretic action of alcohol was no doubt a marked one, but was very frequently neutralised by the cumulative action of the alcohol as a poison. In certain cases it may bring down temperature, but might do so at an inopportune moment, and it certainly exercised some toxic effect on protoplasm which might be deleterious.

Professor Fraser's joke in reference to the plague he would accept as a joke. (Professor Fraser—It was very serious indeed.) He maintained that the conditions under which the Europeans and natives existed in India were very different. The same susceptibility could be induced in animals by starvation, and one was not surprised that the badly fed natives were much more readily affected; and their surroundings were so different from those of the Europeans, that it was entirely ouside the alcohol question.

In connection with Dr Clouston's remarks he would ask if it was worth while to look for pleasure in such epileptic discharges —because they were nothing more than epileptic discharges that took place under the influence of alcohol? Was it worth while running the risks that accompanied the use of alcohol—at any rate for some? As experiments showed, they were no doubt reduced to automata by alcohol—rhythmical movements were produced more readily, and individual movements lost. There was, as it were, a throwing of the balance from single thought movements to automatic movements in a very marked degree. Before leaving this point he would like to ask Professor Fraser what was a useful dose of alcohol? He thought it would be interesting to have from such an authority some indication as to what were to be considered minimum and maximum doses.

Regarding what Dr Ritchie had said about gout and

rheumatism, he thought there was no doubt that alcohol acted in these diseases as a cumulative poison, as does arsenic, and might determine an acute or sub-acute attack.

In reference to the use of alcohol in diphtheria, there was one very peculiar action, even when given in nothing more than very small quantities. It increased the liability to the anti-toxin rash to a remarkable degree. This had been noticed very frequently—it might be part of the beneficial action which it exerts in connection with diphtheria, especially in persons who were otherwise fairly healthy. And in prophylactive cases it was found that rashes occurred in a great number of cases, but if alcohol was not taken at the same time, or for eight or ten days, the rash did not appear in anything like the same proportion.

There were many other points which he could not attempt to enter into. Although he had brought forward for consideration the experiments referred to, he did not say that they answered a great number of the questions which had been raised; but thought they should consider whether the results obtained could be used to throw more light on the subject.

DR J. W. BALLANTYNE (*undelivered remarks*) 'would refer to three only of the many points which might be raised regarding the effect of alcohol upon the immature organism (fœtus or embryo). In the *first* place, it had been clearly shown by the experiments of Féré and others upon the hen's egg that alcohol introduced into the egg during the early hours of incubation interfered very markedly with embryogenesis, producing malformations, monstrosities, and non-developments. It was very noteworthy that the most toxic alcohols were also the most teratogenic, *i.e.* those which when injected into the egg caused the largest proportion of monstrous and anomalous developments. He had met with some cases of malformation in the human subject which suggested to his mind that in their production also alcohol in excess in the parents had, by acting upon the young embryo, been the cause of their production. In the *second* place, he thought it had now been clearly established by the careful analytic experiments of Maurice Nicloux, made both upon animals and upon the human subject, that alcohol taken by the mother passed through the placenta to the fœtus *in utero*. Apparently the transmission was very rapid, a fact which might perhaps account for the negative results obtained by earlier observers, and the amount of alcohol found in the fœtal blood did not fall far short of that in the maternal. He did not think that alcohol reaching the fœtus in this way could be less than harmful, when it was borne in mind that many of the tissues and organs, and more especially the nervous system, were in an incompletely developed state at this period of life.

In the *third* place, it had been shown that the infants born to habitually inebriate women showed a very high degree of mortinatality. In fact it had been proved from prison statistics by Dr W. C. Sullivan that often an alcoholic mother gave birth to her only living and healthy infant after a period of six or seven months' total abstinence within prison walls — a fact which, while it tended to show the evil effects of alcohol upon the unborn infant, also held out a large hope of successful preventive therapeutics.

INDEX

Abscess, psoas, patient exhibited, 48 ; of brain, secondary to an injury to the head, specimen exhibited, 58.
Acne, keloid, case exhibited, 2.
Actinomyces fungus, a microscopical demonstration of, 158.
Affleck, Dr J. O., remarks on gastric hæmorrhage and its treatment, 93 ; remarks on alcohol, 216.
Africa, South, experience with the medical department of the army, original communication by Professor Chiene, C.B., 30; Edinburgh Hospital in, and its work, original communication by Dr F. D. Boyd and Dr George L. Chiene, 63.
Alcohol, in its medical and surgical aspects, original communication by Professor G. Sims Woodhead, 197.
Alexander, Hugh de Maine, M.D., elected a member, 197.
Alopecia areata, several cases exhibited, 157.
Aneurism of aorta, patient exhibited, 43.
Annandale, Professor, original communication, operations for misplaced and undescended testicle, 11; exhibits patient after laminectomy for paralysis, 53.
Apoplexy, cerebral, boy exhibited after being trephined for, 150 ;
Arterio-sclerosis, original communication by Dr William Russell, 132.
Astragalus, fracture of, radiograph exhibited, 57.
Ataxia, Friedrich's, two cases exhibited, 4; locomotor. case exhibited, 19 ; three cases exhibited, 45 ; comparative pathology of, demonstration of, by Dr Chalmers Watson, 62 and 108.
Atrophy, muscular, case exhibited, 146 ; progressive muscular, specimens illustrating, exhibited, 154.
Auricle, casts showing congenital malformation of, exhibited, 154,
Axilla, fatty tumour removed from, of an infant, 59.

Ballantyne, Dr J. W., remarks on alcohol, 229.
Bardswell, Noel Dean, M.D., M.R.C.P. Lond. and Edin., elected a member, 75.
Bath, cabinet, thermal, exhibited, 29.
Bazin's disease, case of, exhibited, 130.
Bell, Dr Joseph, remarks on gastric hæmorrhage and its treatment, 99.
Bladder, hat-pin removed from male bladder, exhibited, 57.
Boyd, Dr Francis D., original communication on the work of the Edinburgh Hospital in South Africa, 63 ; exhibits a patient suffering from pulmonary stenosis, 150.
Brain, tumour of, exhibited, 9 ; four brains showing different seats of cerebral hæmorrhage, exhibited, 24; glioma of, exhibited, 27 ; abscesses of, secondary to an injury of the head, specimen exhibited, 58 ; showing cerebral hæmorrhage, exhibited, 59; showing track of Mauser bullet, exhibited, 151 ; from a case simulating ruptured middle meningeal artery, exhibited, 152 ; specimens illustrating impact of, exhibited, 153.
Brachial plexus, paralysis of, case exhibited, 146.
Bramwell, Dr Byrom, exhibits patients—(1) a case of early tabes, 6 ; (2) cases of locomotor ataxia, 19, 45 ; (3) a peculiar case of muscular atrophy, 146 ; (4) case of syringomyelia, 147 ; (5) case of aneurism of thoracic aorta, 43 ; exhibits specimens—(1) tumour of the right frontal lobe, 9 ; (2) a large glioma of the right motor area, 27 ; (3) a skull from a case of Jacksonian epilepsy, 28 ; remarks on

gastric hæmorrhage and its treatment, 103.
Bramwell, Edwin, M.B., M.R.C.P. Edin. and Lond., elected a member, 1; original communication on myasthenia gravis, 116.
Bruce, Dr Alexander, exhibits patients—(1) a patient after laminectomy for paralysis, 53; (2) a case of paralysis of brachial plexus, 146; (3) a case of sclerosis of the cerebro-spinal type, 146; exhibits for Dr J. O. Affleck a specimen of a greatly dilated heart, 106; exhibits photographs and microscopic specimens illustrating progressive muscular atrophy, sclerosis, and glioma of the Pons Varolii, 154.
Bruce, Dr Lewis C., original communication, clinical and experimental observations upon general paralysis, 171.
Bunting, William Hartley, M.D., F.R.C.S.Ed., elected a member, 2.

Caird, Mr F. M., original communication, operative interference in cases of stricture of large intestine, 158; exhibits patients—(1) four cases of pyloric obstruction treated by gastro-jejunostomy, 61; (2) two patients illustrating certain points in the diagnosis of gastric carcinoma, 148; exhibits specimens—(1) vesical calculi (one half a pound), 154; (2) a large papilloma of the bladder, 154; (3) two small papillomata of the bladder, 154; remarks on gastric hæmorrhage and its treatment, 99.
Calculi, vesical, specimens exhibited, 154; renal, Röntgen photograph of, exhibited, 108.
Cancer of tongue, patient exhibited after removal of, 48.
Carcinoma of skin, case exhibited, 4; of tongue, patient exhibited after removal of, 48; gastric, patients exhibited, 148.
Carmichael, Dr Edward, exhibits a patient with a hypertrophic condition of integuments, 147.
Carmichael, William, M.B., C.M., elected a member, 2.
Carruthers, David Allan, M.B., C.M., elected a member, 61.
Chiene, George Lyall, M.B., C.M., elected a member, 106; original communication, The Edinburgh Hospital in South Africa and its work, 63.
Chiene, Professor John, C.B., original communication, experiences with the medical department of the army in South Africa, 30.
Church, Dr H. M., remarks on alcohol, 225.
Clouston, Dr T. S., remarks on alcohol, 214.
Cotterill, Mr J. M., original communication, case of multiple tumours, 18; exhibits patients—(1) a case of rodent ulcer being treated by X-rays, 151; (2) a case of elbow-joint affection in a hæmophilic patient, 151.
Craig, John, M.B., Ch.B., elected a member, 2.
Cystoscope-catheter, Schlagintweit's exhibited, 131.
Dickie, John Thomas, L.R.C.P. & S. Ed., elected a member, 2.

Dickson, George, M.D., C.M., elected a member, 130.
Diphtheria, larynx from a case of, exhibited, 154.
Drugs, their interference with hepatic metabolism, original communication by Dr Noël Paton and Dr J. Eason, 123.
Drummond, William Blackley, M.B., M.R.C.S.Eng., elected a member, 19.

Eason, John, M.B., M.R.C.P.Ed., elected a member, 2; original communication on a method of estimating the interference with the hepatic metabolism produced by drugs, 123.
Edward VII., King, address to His Majesty by the Society, 106; reply to the Address, 130.
Elbow-joint, affection in a hæmophilic patient, exhibited, 151.
Elder, Dr William, exhibits—(1) four brains showing different seats of cerebral hæmorrhage, 24; (2) dorsal vertebrae with a soft degenerating mass growing from their bodies, 27.
Epilepsy, Jacksonian, skull cap from a case of, exhibited, 28.
Epithelioma, squamous of larynx, patient exhibited after complete extirpation of larynx, 20.

Eye-diseases, pathology of, specimens illustrating, exhibited, 132.

Favus, case of, exhibited, 49.
Femur, fragments of, from the thigh of a Boer, exhibited, 153.
Fibula, fracture of, specimen exhibited, 57.
Food, regurgitation of, case exhibited, 5.
Forceps, laryngeal, exhibited, 158.
Fordyce, Alexander Dingwall, M.B., Ch.B., elected a member, 130.
Foulis, Dr James, remarks on alcohol, 224.
Fracture of fibula, specimen exhibited, 57; of humerus, radiograph exhibited, 57; of astragalus, radiograph exhibited, 57; of malleolus, specimen exhibited, 57.
Fraser, Professor T. R., remarks on alcohol, 210, 226.
Friedrich's ataxia, two cases exhibited, 4.
Fungus, actinomyces, microscopical demonstration of, 158.

Gairdner, Sir William, remarks on gastric hæmorrhage and its treatment, 97.
Gardiner, Frederick, M.B., C.M., elected a member, 1.
Gastric ulcer, perforating, patient exhibited after operation for, 49.
Gastro-jejunostomy, four cases treated by, for pyloric obstruction, exhibited, 61.
Gibson, Dr G. A., exhibits two brothers suffering from Friedrich's hereditary ataxia, 4.
Glioma, large, of brain, exhibited, 27; of pons varolii, exhibited, 154.
Gluteus maximus, patient exhibited with paralysis of, 52.
Greenfield, Professor, remarks on gastric hæmorrhage and its treatment, 98.
Gulland, Dr G. L., gives a microscopical demonstration of specimens of actinomyces fungus, 158.

Hæmorrhage, cerebral, four brains showing different seats of, exhibited, 24; brain exhibited, 59; gastric, and its surgical treatment, original communication by Mr Mayo Robson, 76.
Hat-pin, 3½ inches long, removed from male bladder, exhibited, 57.
Heart dilated, exhibited, 106.
Hernia, strangulated, stenosis of small intestine after, original communication by Mr Alexis Thomson, 110.
Hip-joint, amputation through, patient exhibited, 22.
Hospital, Edinburgh, in South Africa, and its work, original communication by Dr F. D. Boyd and Dr G. L. Chiene, 63.
Humerus, fracture of, radiograph exhibited, 57.
Hypertonus, arterial, original communication by Dr William Russell, 132.

Integument, hypertrophic condition of, patient exhibited, 147.
Intestine, large, operative interference in cases of stricture of, original communication by Mr F. M. Caird, 158.
Intestines, transverse rupture of, specimen exhibited, 60; stenosis of, after strangulated hernia, original communication by Mr Alexis Thomson, 110; from a case of idiopathic distension of the colon, exhibited, 154.
Intussusception, an infant exhibited after operation for, 51.

Jacksonian epilepsy, skull cap from a case of, exhibited, 28.
James, Dr Alexander, remarks on gastric hæmorrhage and its treatment, 102.
Jamieson, Dr Allan, exhibits patients—(1) case of acne keloid, 2; (2) case of tylosis palmæ et plantæ, 3; (3) case of carcinoma of skin, 4; exhibited microscopic specimens of cervical tumours, 56.
Jamieson, John Boyd, M.B., F.R.C.S.Ed., elected a member, 2.
Johnston, Dr M'Kenzie, exhibits patients—(1) a patient (for Mr Alexis Thomson) two months after complete removal of larynx, 20; (2) a boy after removal of a laryngeal papilloma, 156; (3) a patient with a lympho-sarcoma of tonsil, 42; exhibits a laryngeal forceps, 158.

Ker, Dr Claud B., remarks on alcohol, 225.
Knee-joint, cartilaginous loose bodies from, exhibited, 107.
King Edward VII., Address to His

Majesty by the Society, 106; reply to the Address, 130.

Laminectomy for paralysis, patient exhibited after operation, 53.
Langwill, Dr H. G., exhibits a case of regurgitation of food, 5.
Larynx, extirpation of, for epithelioma, patient exhibited after operation, 20; from a case of diphtheria, exhibited, 154.
Lawson, David, M.D., elected a member, 61.
Leech, Joseph William, M.D., elected a member, 155.
Littlejohn, Dr Harvey, exhibits specimens—(1) brain showing cerebral hæmorrhage, 59; (2) cut-throat, 59; (3) a specimen showing complete transverse rupture of the small intestine, also of the mesentery, 60; (4) a double uterus, 60; (5) a stomach showing acute gastritis, 60.
Livingston, George Robert, M.B., C.M., elected a member, 130.
Luke, Thomas Davy, M.B., Ch.B., elected a member, 1.
Lupus, treated by X-rays, patient exhibited, 131.
Lymphoma, patient exhibited after removal of, 21.
Lympho-sarcoma of tonsil, patient exhibited, 42.

M'Allum, Donald C. A., M.B., C.M., elected a member, 19.
M'Donald, Stuart, M.B., M.R.C.P.Ed., elected a member, 75.
MacGillivray, Dr C. W., remarks on gastric hæmorrhage and its treatment, 101.
Mackenzie, Dr W. Leslie, remarks on alcohol, 217.
M'Laren, Mr J. Shaw, exhibits patients—(1) a patient after removal of the tongue for cancer, 48; (2) a psoas abscess in a girl aged 16, 48; (3) a case of tuberculous disease of the wrist, 48; exhibits specimens—(1) a recent Pott's (?) fracture, 57; (2) Schlagintweit's catheter-cystoscope, 131; (3) radiograph of fractured condyle of humerus, 57; (4) radiograph of the astragalus, 57.
M'Larty, Malcolm, M.B., C.M., elected a member, 2.
Malleolus, fracture of, specimen exhibited, 57.
Matthew, Edwin, M.B., C.M., elected a member, 19.
Mauser-bullet, brain showing track of, exhibited, 151; impact of, specimens exhibited, 151.
Meningocele, patient exhibited with large, 51.
Mesentery, transverse rupture of, specimen exhibited, 60.
Miller, Mr A. Gordon, reference to the death of Her Majesty, 75.
Moorhouse, James Ernest, M.D., C.M., elected a member, 61.
Morphœa, case of, exhibited, 48.
Myasthenia gravis, original communication by Dr Edwin Bramwell, 116.

Nicholson, H. Oliphant, M.B., elected a member, 1.

Office-bearers, 1.

Papilloma, of larynx, patient exhibited after removal of, 156; of bladder, specimens exhibited, 154.
Paralysis, of brachial plexus, case exhibited, 146; musculo-spiral, after operation, patient exhibited, 155; of gluteus maximus, patient exhibited, 152; laminectomy for, patient exhibited, 53.
Paralysis, general, clinical and experimental observations upon, original communication by Dr Lewis C. Bruce, 171; observations on the pathogenesis of, original communication by Dr W. Ford Robertson, 180.
Paterson, James Veitch, M.B., F.R.C.S.Ed., elected a member, 19; exhibits a series of naked-eye specimens, mounted in glycerine jelly, illustrating the pathology of eye diseases, 132.
Paton, Dr Noël, original communication on a method of estimating the interference with the hepatic metabolism produced by drugs, 123.
Prostate, enlarged, specimen exhibited, 9.
Purves, Dr Robert B., exhibits a case of musculo-spiral paralysis, after operation, 155.
Pyloric obstruction, four cases of, treated by gastro-jejunostomy exhibited, 61.

Queen Victoria, reference to her death by the President, 75.

Ringworm, treated by X-rays, patient exhibited, 156.
Ritchie, Dr James, remarks on alcohol, 222.
Ritchie, William Thomas, M.D., M.R.C.P.Ed., elected a member, 19.
Robertson, Dr W. Ford, original communication on the pathogenesis of general paralysis of the insane, 180.
Robson, Mr A. W. Mayo, F.R.C.S., original communication on gastric hæmorrhage and its surgical treatment, 76.
Röntgen apparatus, improved, demonstrated, 63.
Russell, Dr William, original communication, arterial hypertonus and arterio-sclerosis, their relations and significance, 132.
Rutherford, James Mair, M.B., C.M., elected a member, 19.

Sarcoma of testicle from an infant, exhibited, 155.
Scalp, complete evulsion of, case exhibited, 50.
Schlagintweit's catheter-cystoscope, exhibited, 131.
Sclerosis, of the cerebro-spinal type, case exhibited, 146; amyotrophic lateral, specimens illustrating, exhibited, 154.
Sillar, William Cameron, M.B., B.Sc., elected a member, 2.
Simpson, G. F. Barbour, M.B., Ch.B., elected a member, 2.
Sinclair, Arthur Hy. Havens, M.D., F.R.C.S.Ed., elected a member, 2.
Skin, carcinoma of, case exhibited, 4.
Skin-grafting, patient exhibited, showing result of, 50.
Skirving, Dr Scot, C.M.G., exhibits specimens—(1) brain, showing track of mauser bullet, 151; (2) brain from a case simulating ruptured middle meningeal artery, 152; (3) specimens illustrating bullet impact, 153; (4) stomach of a patient eight days after operation for perforated gastric ulcer, 153; (5) large perineal tumour, 153; (6) fragments of femur from the thigh of a Boer, 153; (7) twisted thimble or sheath of mauser bullet, 153.
Skull cap, from a case of Jacksonian epilepsy, exhibited, 28.
Small intestine, stenosis of, after strangulated hernia, original communication by Mr Alexis Thomson, 110; enterectomy for stricture of, patient exhibited, 149.
Stenosis, of small intestine after strangulated hernia, original communication by Mr Alexis Thomson, 110; pulmonary, patient exhibited, 150.
Steven, John William, M.B., F.R.C.S. Ed., elected a member, 1.
Stevenson, John, M.B., F.R.C.S.Ed., elected a member, 1.
Stiles, Mr H. J., exhibits patients—(1) case of paralysis of gluteus maximus, 52; (2) case of complete evulsion of the scalp, 50; (3) a large occipital meningocele, 51; (4) a boy who had been trephined for cerebral apoplexy, 150; (5) a boy after removal of a lymphoma from the neck, 21; (6) a patient after operation for perforated gastric ulcer, 49; (7) an infant after operation for intussusception, 51; (8) a girl after amputation through hip-joint, 22; exhibits specimens—(1) brain showing secondary abscesses, 58; (2) congenital fatty tumour from axilla of an infant, 59; (3) a sarcoma of the testicle of an infant, 155; (4) a portion of hat-pin ($3\frac{1}{2}$ inches) removed from the male bladder, 57; (5) intestines from a girl who had suffered from idiopathic distension of the colon, 154.
Stomach, showing acute gastritis exhibited, 60; eight days after operation for ruptured gastric ulcer, exhibited, 153.
Syringe, a glass-barrelled, exhibited, 154.
Syringomyelia, case of, exhibited, 147.

Tabes, case of, exhibited, 6.
Testicle, misplaced and undescended, original communication by Professor Annandale, 11; sarcoma of, in an infant, specimen exhibited, 155.
Thompson, John Andrew Douglas, M.B., Ch.B., elected a member 130.
Thomson, Mr Alexis, original communication—stenosis of the small

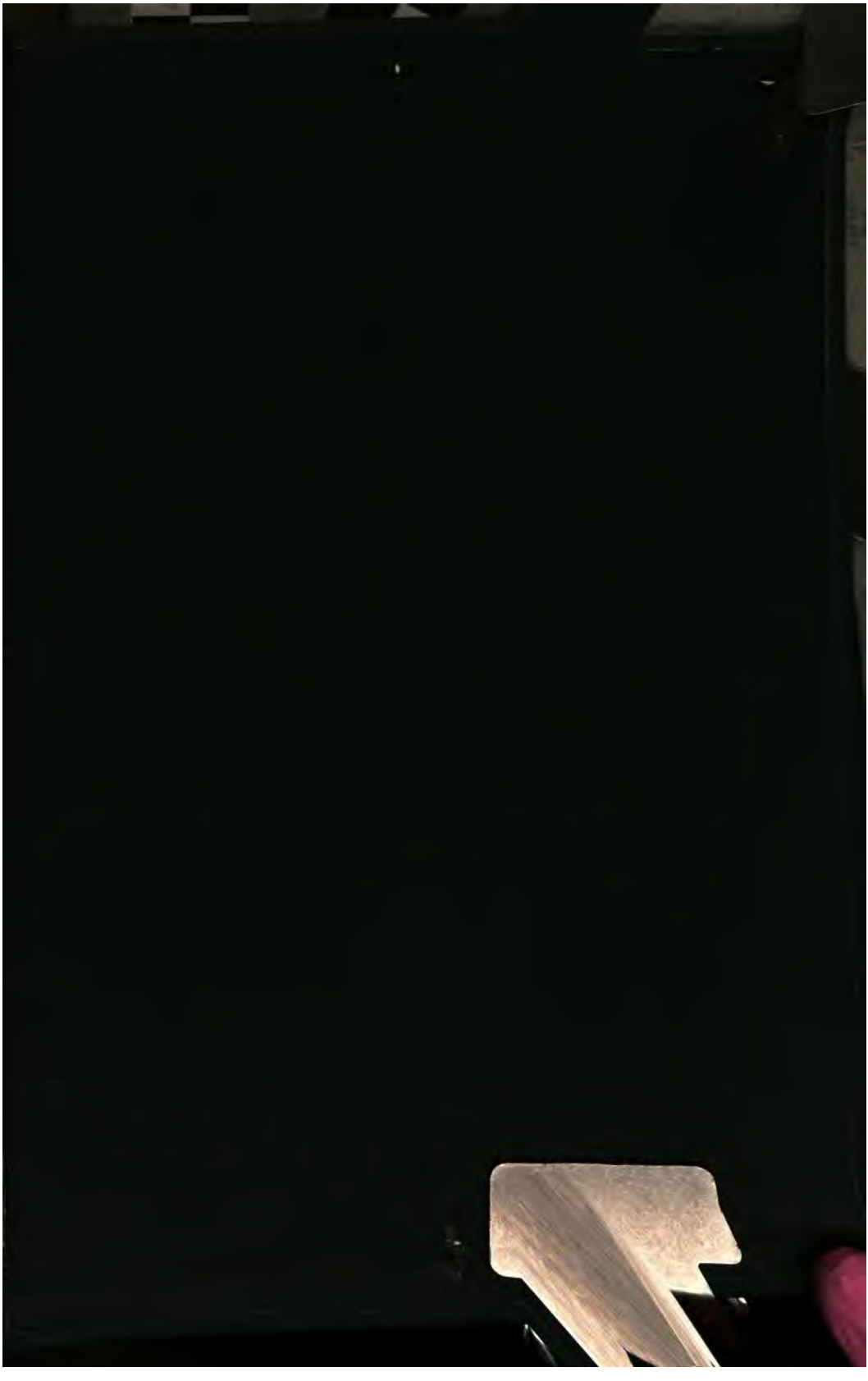
intestine after strangulated hernia, 110; exhibits patients—(1) a patient after enterectomy, 149; (2) a patient after complete extirpation of larynx, 20; (3) a patient after removal of middle two-thirds of tibia, 47; exhibits specimens—(1) multiple cartilaginous loose bodies from the knee-joint, 107; (2) a deformed shaft of a tibia, 56; exhibits a glass-barrelled syringe, suitable for irrigating or aspirating, 154.
Thomson, H. Torrance, M.D., C.M., elected a member, 2.
Throat-cut, specimen exhibited, 59.
Tibia, removal of middle two-thirds of; patient exhibited, 47; a deformed, exhibited, 56.
Tongue, cancer of, patient exhibited after removal of tongue, 48.
Tonsil, tumour of, patient exhibited, 42.
Tuberculous disease of wrist, patient exhibited, 48.
Tumour, of brain, exhibited, 9; lymphoma of neck, patient exhibited after removal of, 21; of dorsal vertebræ, exhibited, 27; of tonsil, patient exhibited, 42; cervical, microscopic specimens of, exhibited, 56; fatty, from axilla of an infant, exhibited, 59; large perineal, exhibited, 153.
Tumours, resection of, original communication by Mr F. M. Caird, 158; multiple of unusual type, original communication by Mr J. M. Cotterill and Dr D. A. Welsh, 18.
Turner, Dr Dawson, demonstrates an improved Röntgen apparatus, 63; exhibits Röntgen photograph of renal calculi, 108.
Turner, Dr Logan, exhibited—(1) a larynx from a case of primary laryngeal diphtheria, 154; (2) two casts showing congenital malformation of the auricle, 154.
Tylosis, palmæ et plantæ, case exhibited, 3.

Ulcer, gastric, perforating, patient exhibited after operation for, 49; stomach exhibited eight days after operation for, 153; rodent, treated by X-rays, patients exhibited, 131 and 151.
Uterus, double, specimen exhibited, 60.

Vertebræ, dorsal, with tumour, exhibited, 27.
Victoria, Queen, reference to her death, by the President, 75.

Walker, Dr Norman, exhibits patients—(1) a case of morphœa, 48; (2) a case of favus, 49; (3) a case of Bazin's disease, 130; (4) case of lupus being treated by X-rays, 131; (5) a case of rodent ulcer, being treated by X-rays, 131; (6) a case of ringworm being treated by X-rays, 156; (7) four cases of alopecia areata, 157.
Wallace, Mr David, C.M.G., exhibits a specimen of enlarged prostate, 9; remarks on gastric hæmorrhage and its treatment, 101.
Waterston, David, M.D., F.R.C.S.Ed., elected a member, 2.
Watson, Dr Chalmers, exhibits a thermal bath cabinet, 29; gives demonstrations on the comparative pathology of ataxia, 62, 108.
Welsh, David Arthur, M.D., M.R.C.P.Ed., elected a member, 2; original communication on a case of multiple tumours of unusual type, 18; remarks on alcohol, 224.
Wilson, James, M.B., C.M., elected a member, 2.
Woodhead, Professor G. Sims, original communication, notes on alcohol in its medical and scientific aspects, 197, 227.
Wrist, tuberculous disease of, patient exhibited, 48.

X-rays, in treatment of ringworm by, patient exhibited, 156; treatment of lupus by, patient exhibited, 131; treatment of rodent ulcer by, patients exhibited, 131, 151.

Young, Dr P. A., remarks on alcohol, 224.

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